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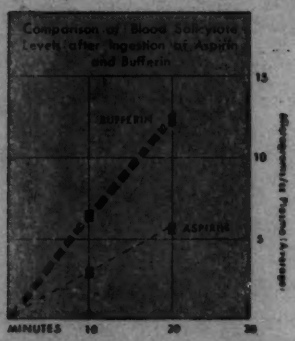

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JOURNAL OF ORAL SURGERY

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VOLUME 15 • JANUARY 1957 • NUMBER 1 JOURNAL OF ORAL SURGERY

Trauma as a factor causing delayed repair of dental extraction sites

■ *Charles C. Alling,* D.D.S., M.S., El Paso, Texas, and Donald A. Kerr,† D.D.S., M.S., Ann Arbor, Michigan*

One of the ever-present problems facing the dentist who extracts teeth is the complication characterized by failure of repair of the alveolus and persistent attendant pain. The condition is commonly referred to as "dry socket." Because of the frequency with which this condition occurs, there has been much interest in the problem and many suggestions as to its cause. One of us has observed that the teeth's having a gross amount of periodontal membrane adherent after extraction predisposes to the development of alveolar osteitis. It has also been observed that extensive and vigorous use of elevators during dental extraction results in a high incidence of dry sockets. It therefore seemed logical that if trauma at the time of extraction destroyed or severely damaged the periodontal membrane, organization of the blood clot would not occur, the alveolar bone would lose its blood supply and become necrotic, and healing would be delayed.

The preceding observations and rationalization initiated this study. Monkeys were selected as the experimental animal because of the close similarity of their dentition to the human dentition.

■ *Review of the Literature*

The term dry socket is in popular usage and acceptance, although it does not describe accurately the process involved. The phrases dry socket,[1-5] necrotic alveolar socket,[6] painful socket, or Archer's term alveolalgia,[5,7-9] and the more accurate term

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†Professor of oral pathology and periodontia, School of Dentistry, University of Michigan.

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8. Orlean, S. L. Postoperative pain and its control by the general practitioner. D. Cosmos 78:743 July 1936.

9. Schroff, J., and Bartels, H. A. Treatment of painful sockets with sodium perborate. (Abst.) J. D. Res. 9:81 Jan. 1929.

alveolar osteitis are seen.[10,11] The concept is that of a tooth socket in which the blood clot is disintegrated and is attended by a severe neuralgia, a foul taste and odor, with or without a purulent exudate, and with or without infection. There is a general recognition that regardless of etiology the process is a localized osteomyelitis or osteitis.[12-15] Not only have different terms been developed but also there is a variance of conclusions as to the etiology of delayed repair of extraction sockets. Most writers believe that a combination of factors is possible but conclude that one agent or another is paramount.

Trauma was nearly a consistent observation in the literature as a predisposing cause of delay in repair or was generally listed as a cause of postoperative pain after tooth extraction.[6,16-27] Machat[27] had listed 15 causes for postextraction pain but finally lodged on one factor as outstanding—trauma. Pell[3] wrote that extraction of teeth from sockets with atrophic periodontal membranes caused excessive trauma to the bony walls of the sockets and predisposed the wound to delayed repair. From vast clinical experience, Harang[28] concluded that a burnishing type of trauma as incurred by undue manipulation of a tooth during extraction predisposed to a delay in repair.

That trauma to an area during extraction of a tooth causes a local release of histamine which blocks the physiologic actions of adjacent cells and results in a delay in repair was reported by Silverman[29] in a preliminary report of a clinical investigation. Wald[30] described the traumatized socket as being more susceptible to infection by microorganisms. Podolin[12] and Armstrong,[17] however, did not feel that there was justification for assuming that a traumatized socket was an etiologic factor in dry sockets; and Archer[7] indicated that trauma did not play as important a role as had been described in past writing, although 56 per cent of the patients with painful sockets in his series had a history of trauma during extraction.

The need for an adequate blood supply for repair of a dental extraction site receives considerable attention in the literature. Sclerosis of alveolar bone or a dense lamina dura prior to the extraction

10. Elwell, K. R. New approach to the treatment of dry socket. J.A.D.A. 31:615 May 1944.

11. Russell, L. T., Jr. Comparison in methods of prevention of local alveolar osteitis. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 30:95 Feb. 1944.

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13. Schram, W. R. Theoretical considerations of bone and tissue healing in relation to oral surgery. Northwest. Univ. Bul. 47:4 Winter Quart. 1947.

14. Silverman, M. Pain. D. Cosmos 77:1211 Dec. 1935.

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22. Eman, V. H. Dry socket problem. J. Oral Surg. 2:158 April 1944.

23. Kemper, J. W. Extraction of teeth and other surgical procedures. In American textbook of operative dentistry, Ward, M. L., ed. 7. Philadelphia, Lea & Febiger, 1940, p. 835.

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26. Lyons, C. J. Extraction of teeth and other surgical procedures. In American textbook of operative dentistry, Ward, M. L., ed. 6. Philadelphia, Lea & Febiger, 1926.

27. Machat, B. B. Facility of surgery in the general practice of dentistry. D. Cosmos 76:342 March 1934.

28. Harang, H. L. Prevention of dry sockets in extraction of teeth. Oral Surg., Oral Med. & Oral Path. 1:601 July 1948.

29. Silverman, R. E. Use of antihistamines in oral surgery: a preliminary report. J. Oral Surg. 11:231 July 1953.

30. Wald, A. Postoperative complications in exodontia: their treatment and prevention. D. Cosmos 74:72 Jan. 1932.

is recognized as limiting the amount of blood flow to the area after extraction.[3,31-34] One of Schram's[13] conclusions, that any procedure interfering with the blood supply would produce poor healing, is in accord with Christopher's[19] statement that the blood vessels when unable to expand in the bony wall of the dental alveolus because of inflammatory phenomena, become thrombosed and a loss of blood supply results. Schwarz[35] mentioned that the poor blood supply causes a local necrotic condition in which angioblastic or fibroblastic tissues cannot proliferate; consequently, the blood clot is not organized and healing is further delayed.

The invasion of the alveolus by microorganisms after dental extraction with a resulting delay in repair has been greatly emphasized in the literature.[5,8,10,12,13,15,36,37] The work of Schroff and Bartels[9] is interesting in that they found anaerobic organisms, fusiform bacilli and spirochetes in painful sockets and that on the cessation of pain these organisms were no longer obtainable from the affected sockets. Belding and Belding[31] found a fusospirochetal complex of organisms as the most common postextraction infection of sockets. Archer[7] wrote that 80 per cent of his series of painful sockets had either diplostreptococci or streptococci. He indicated that these organisms were the most common etiologic agents of postextraction pain. Claflin[20] was able to delay healing in tooth sockets of dogs by introduction of staphylococci and streptococci from infected human sockets. Grandstaff,[38] however, reported that the bacteriologic findings in 20 instances of normally healing sockets and in 20 instances of delayed or abnormally healing sockets were not significantly different.

■ *Methods and Experimental Data*

Because their structure and function resemble that of human beings, monkeys were the laboratory animals of choice to demonstrate the effect of a burnishing trauma to the dental alveolar walls. Rhesus monkeys were used; they averaged about eight months of age. They were noninfected, apparently healthy, animals.

Different anesthetic agents, such as intravenous 2.5 per cent solution of thiopental sodium, open drop ether and insufflation ether, were employed, but the most satisfactory anesthetic regimen consisted of intramuscular injections of sodium pentobarbital. The latter anesthetic solution consisted of 75 mg. of sodium pentobarbital dissolved in 1 cc. of 60 per cent solution of propylene glycol. The 60 per cent glycol solution was made by diluting 100 per cent propylene glycol with 95 per cent alcohol. One cubic centimeter, that is, 75 mg. of sodium pentobarbital was injected intramuscularly 30 minutes preoperatively; this provided satisfactory control of the animals for the extraction procedures. In a few instances it was

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32. Hudack, S. S. Clinical basis for bone healing. J. Periodont. 19:7 Jan. 1948.

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35. Schwarz, H. J. Question of the dry socket. J.A.D.A. 20:1502 Aug. 1933.

36. Sicher, H. Some principles of bone pathology. J. Oral Surg. 7:104 April 1949.

37. Winter, Leo. Local anesthesia and exodontia. D. Cosmos 73:545 Jan 1931.

38. Grandstaff, C. H. Influence of the oral bacteria on the healing of exodontia wounds. (Abst.) Bur 35:90 July 1935.

necessary to supplement the sodium pentobarbital with inhalation ether, and it was most convenient to do this with insufflation.

From 25 monkeys the right mandibular first molar was extracted with forceps, and the left mandibular first molar was removed with straight elevators. The socket walls of the left first molar were burnished thoroughly with a metallic instrument. No dressings were used in the sockets, and no attempt was made to control artificially the hemorrhage from the alveolar sockets. Each animal provided its own control inasmuch as the traumatized socket and the simple extraction socket were submitted to the same environmental, systemic and local factors.

The sockets were examined clinically and histopathologically. A fairly uniform set of observations was possible for all operative sites after the fifth postoperative day. No delay clinically was inferred when a mucosal covering of the extraction site was evident with no acute inflammation. Slight to moderate delay in repair was noted clinically when a bony sequestrum or an area of granulation tissue was visible at the extraction site and the socket was otherwise covered by mucosa. Pronounced delay in repair was the sign of an acute alveolar osteitis seen in human beings, that is, an open socket with little or no reparative tissues grossly present.

The clinical examination of the one monkey sacrificed the first day after extraction disclosed no substantial difference between the left and right sides on clinical examination. The histopathologic examination showed focal areas of fibroblastic activity and periodontal membrane completely lining the alveolus of the right (control) side, whereas the left (affected) alveolus had less residual periodontal membrane and no fibroblastic activity.

The three monkeys sacrificed at one week (two at seven days, one at six days) showed clinically normal repair of the control (right) alveolus, whereas the affected (left) alveolus disclosed a moderate delay in each instance. Histopathologically, the right alveoli all showed retention and early organization of the clot with slight to abundant periodontal membrane. The left sides had partially retained blood clots, abundant exudate and slight or no evidence of residual periodontal membrane (Fig. 1).

The 20 monkeys sacrificed at two weeks each had normal repair clinically evident in the control (right) alveoli. On the traumatized (left) side, 3 alveoli had normal repair, 11 had moderate delay, and 6 had a pronounced delay in repair (Fig. 2). The histopathologic findings reflected the clinical impressions: The affected sites showed delay in repair, as compared to the control sites, by having varying degrees of incomplete epithelization, less bone formation, sometimes surface sequestra surrounded by colonies of organisms, and varying degrees of fibrosis of surrounding marrow spaces (Fig. 3). On the affected sides, it was noted frequently that the tooth anterior to the extraction site showed effects of trauma induced by the elevator. The periodontal tissues of the anteriorly placed tooth were infiltrated with inflammatory cells, and there was active resorption of the alveolar bone.

The one monkey sacrificed at three weeks demonstrated a normally repairing socket on the control (right) side and a noticeable delay in repair on the affected

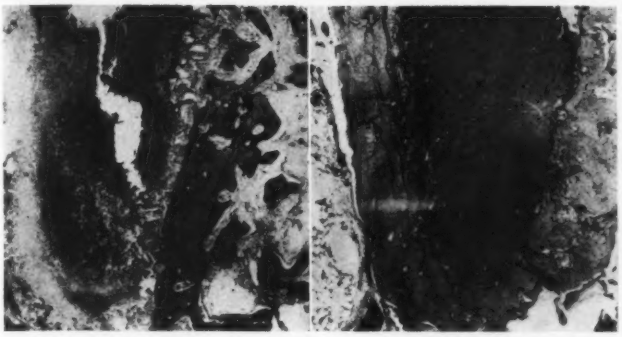
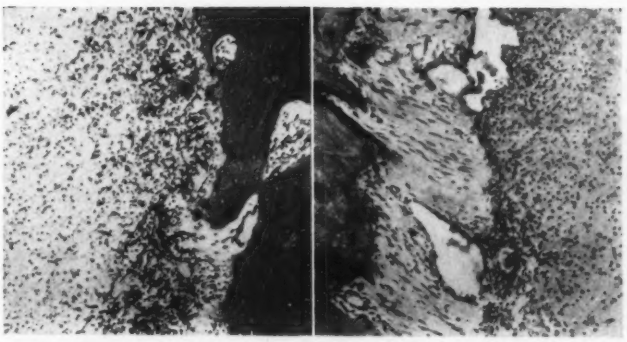
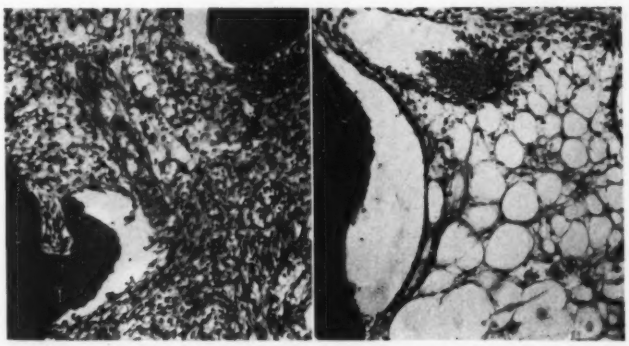


Fig. 1—Monkey B. One week postextraction. Clinical impression was that this demonstrated moderate delay in healing on traumatized side. Left: Traumatized socket. Residual periodontal membrane minimal. Blood clot only partially retained. Right: Control socket. Blood clot completely fills socket. Residual periodontal membrane about alveolus has started to organize clot



High power view of same monkey, one week postextraction. Left: Traumatized socket. Only few fibers of periodontal membrane present. Hemolysis of blood cells leaving delicate fibrin strands of partially liquefied clot. No evidence of organization. Right: Control side. Abundant periodontal membrane still arranged as principal groups of fibers. No evidence of liquefaction of clot. Early organization



High power view of marrow spaces adjacent to alveolus of same monkey, one week after extraction. Left: Traumatized side. Subacute osteitis with fatty marrow infiltrated by inflammatory cells and early proliferating fibroblasts. Right: Control side. Marrow essentially normal with only a few scattered inflammatory cells

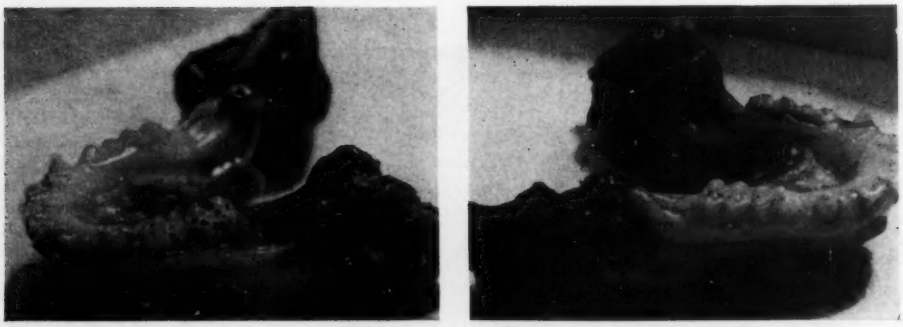


Fig. 2—Monkey F. Two weeks' postextraction. Clinical appearance of socket. Left: Traumatized socket. Alveolus appears free of blood clot, shows no reduction in size and no evidence of epithelial regeneration. Right: Control side. Blood clot present, reduction in size of alveolus and some epithelial regeneration

(left) side. The microscopic findings were similar to those of the two-week specimens, the affected side showing incomplete epithelization and less new bone formation.

■ *Discussion*

The microscopic findings in the control sockets were fairly constant. Nearly all showed partial to almost complete regeneration of surface epithelium. In those instances in which the regeneration was complete, there was a minimal inflammatory response in the subepithelial connective tissue. The alveolar bone showed regeneration from all surfaces of the alveolus and at the alveolar crest. In most instances this filled from three fourths to all of the radicular area. The defect in cortical bone at the crest was filled with maturing connective tissue with minimal evidence of inflammatory infiltrations. Occasionally, there was a small fragment of dentin and cementum present which was surrounded by proliferating fibrous tissue. It had produced no giant cell response and was not acting as a foreign body, so there was no effect on healing.

In those instances in which epithelization was incomplete there were numerous inflammatory cells at the border of the open area. These were closely limited to the surface. In these instances the amount of new bone formation was less. Only about two thirds of the alveolus was filled with new bone, and the amount at the alveolar crest was less than in those sockets completely epithelized. Occasional small sequestra of bone were noted in two instances. They were near the surface and were completely surrounded by epithelium. They did not appear to delay the healing procedure. There was minimal change in the marrow spaces surrounding the alveolus. Occasional small inflammatory foci were observed near the alveolar plate where it was perforated by vessels.

The sockets which were traumatized showed only slight to partial epithelization. The opening of the alveolus was partially devoid of blood clot or organized

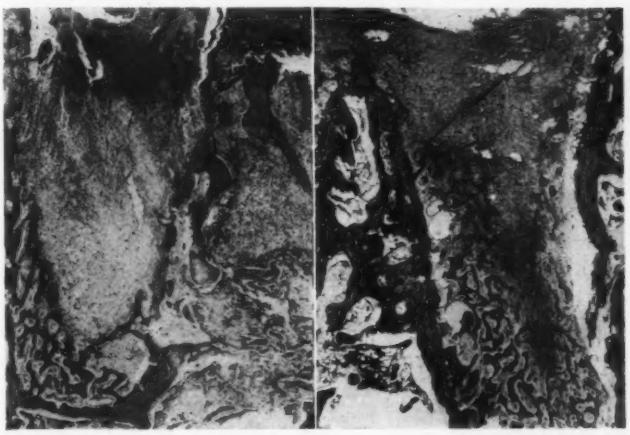
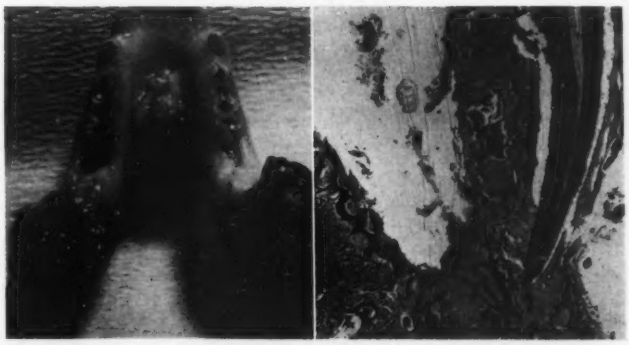
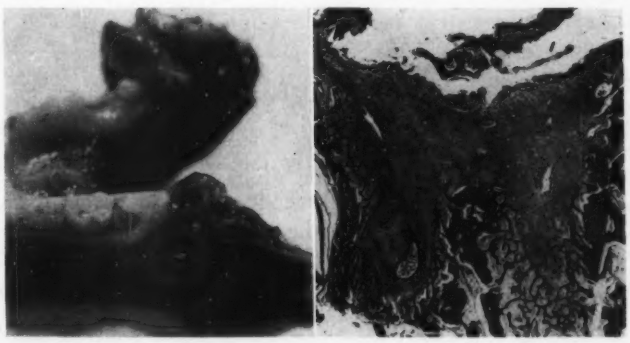


Fig. 3—Monkey F. Two weeks' postextraction. Left: Traumatized side. Alveolus partially filled with partially liquefied clot. At top of alveolus, mass of cell debris and colonies of mouth organisms. Some regeneration of bone in apicolateral area. Fragmentation of alveolar bone. Severe subacute osteitis completely surrounds alveolus. Right: Control socket. Partially organized blood clot completely fills alveolus. New bone formation evident in apical area and from nearly all lateral borders of alveolus. This reaches almost to level of intraradicular septa at right side of picture. No evidence of osteitis around socket



Left: Monkey G. Two weeks' postextraction. Left socket is free of blood clot and shows no evidence of healing whereas right side shows normal repair. Right: Traumatized socket showing absence of blood clot. Necrosis of alveolar bone and interradicular septa on mesial surface. New bone formation outside necrotic alveolus. Note vegetable debris in alveolus



Monkey S. Two weeks' postextraction. Left: Sequestrum protruding from traumatized socket on left side. Right: Alveolus shown at left. No epithelization of surface. Sequestrum surrounded by colonies of organisms on surface. Partial organization of blood clot with new bone in apical third of mesial root. Fracture in midportion of mesial alveolar wall. Epithelial proliferation surrounding debris in apex of distal part of alveolus

tissue. Many contained varying sized pieces of dead bone surrounded by colonies of mouth organisms and food debris. In some instances the food debris and organisms could be seen in the radicular portion of the alveolus. The alveolar bone lining the alveolus in some instances was fractured and entirely necrotic but still partially attached. There was minimal evidence of periodontal membrane. When present it was in the apical one fourth of the alveolus. New bone formation followed the same pattern as the residual periodontal membrane. In most instances it was limited to the apical one fourth or one third and did not extend to or over the alveolar crest.

The marrow spaces surrounding the alveolus demonstrated replacement of the normal cellular marrow by a young proliferating fibrous connective tissue heavily infiltrated with leukocytes. In many sections this reaction extended for some distance from the alveolus. In some of the marrow spaces there was evidence of giant cell formation of osteoclastic type which indicated early destruction and separation of the necrotic alveolar bone.

When the control and experimental alveoli are compared, it is evident that trauma has produced delay in healing. In those alveoli traumatized, the evidence of repair was in the apical one fourth where the periodontal membrane was not destroyed because of the small size of the alveolus, whereas in the more spacious areas where the periodontal membrane was destroyed organization of a clot and regeneration were absent. This indicates the importance of the periodontal membrane in organization and, therefore, in retention of the clot and, if the blood clot becomes organized, with the formation of granulation tissue. The periodontal membrane is protective against the ingress of organisms, and it is our impression that their presence in the alveolus is of no significance in producing osteitis. All alveoli contain organisms of the oral flora but apparently they are only of significance when the clot is unorganized. In a tooth with periapical inflammation or infection, there may be destruction of the periodontal membrane with failure to organize the apical portion of the clot and thus delay in healing. It is our interpretation, however, that it is the absence of periodontal membrane rather than the presence of inflammation which is responsible for the dry socket.

■ *Summary*

1. The lower right first molars of 25 monkeys were extracted by forcep technic. The corresponding left first mandibular molars were extracted with straight elevators and the alveolus burnished with a blunt instrument to destroy the periodontal membrane.

2. The animals were sacrificed at 1, 7, 14 or 21 days after extraction.

3. The control side and the traumatized side were compared clinically at the time of sacrifice to determine the delay in healing. Histologic studies also were made of both control and traumatized sockets to compare the rate of healing.

4. Clinically there was no evidence in delay of healing in any of the control sockets.

5. Clinical examination of the traumatized sockets showed no delay in healing in 4 animals, moderate delay in heal-

ing in 14 animals and pronounced delay in healing in 7 animals.

6. Microscopic examination of the control sockets demonstrated an intact blood clot being organized for the residual periodontal membrane.

7. Microscopic examination of the traumatized sockets revealed varying degrees of failure of organization of the clot. The degree was proportional to the absence of periodontal membrane.

8. Traumatized sockets demonstrated intense subacute osteitis surrounding the alveoli. This was absent in the control animals.

■ *Conclusions*

Burnishing of the alveolus produces destruction of the periodontal membrane. Delay of healing in dental sockets is correlated with the amount of periodontal membrane residual after extraction. The absence of periodontal membrane prevents organization of the blood clot, which sloughs out, permitting the invasion of organisms and the entrapment of debris. This condition plus the attendant trauma to the alveolar bone results in an osteitis responsible for the pain and delay of healing, which is a characteristic clinical observation in dry socket.

Role of tantalum in reconstruction of orbitomalar and microgenic deformities

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The element tantalum (anatomic weight 180.88; anatomic number 73) was discovered by the Swedish scientist Ekeburg in 1802 and seems to satisfy most of the requirements for the surgical use of an imbedded metal. Tantalum is similar to steel in color, strength and ductility, but it is twice as dense and becomes fatigued at a much slower rate. Tantalum is comparable to glass in its resistance to the body chemistry and is resistant to most acids, being acted on only by hydrofluoric acid, concentrated sulfuric acid and concentrated, strong alkalies and their salts. Burke[1] and Carney,[2] the first to make surgical use of tantalum, believed it to be a physiologically inert metal. This opinion has since been confirmed by

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1. Burke, G. L. Corrosion of metals in tissues; and introduction to tantalum. Canad.M.A.J. 43:125 Aug. 1940

2. Carney, H. M. Experimental study of tantalum. Proc. Soc. Exper. Biol. & Med. 51:147 Oct. 1942.

Pudenz,[3] Craig,[4] Hook[5] and others. Tantalum is apparently resistant to corrosion because its oxide, which is thin and transparent, is also strong and tenacious and forms a protective film over the metal. The density of bone in contact with tantalum increases and the adjacent periosteum becomes thickened. The tantalum itself becomes surrounded by a thin fibrous envelope or capsule.

Tantalum has been used primarily in cranioplasties, for which purpose it is particularly satisfactory as the plate can be contoured easily in advance and then precisely fitted to the defect at the time of surgery. In this manner surgical exposure of the defect to obtain an impression from which some type of casting is to be made can be avoided. Several simple technics for the fabrication of tantalum skull plates have been described in the literature,[6,7] and probably hundreds of tantalum cranioplasties have been performed. Unfortunately, the ease with which tantalum can be wrought and the relative availability of the required equipment have made it possible for poorly contoured, ill-fitting plates to be prepared, many of which had to be removed because of marginal irritation, inadequate anchorage with slipping, poor cosmetic results or because there were insufficient perforations to permit the exchange of fluids.

In 1947, Lane and Webster[8] surveyed all the tantalum cranioplasties which had been performed up to that date in 115 Veterans' Administration hospitals and found that only 52 plates had had to be removed. Of this number, one half involved the frontal area and one third included the frontal sinuses. Many neurosurgeons believe that if the frontal sinuses are involved, the lining membrane should be curetted first, and the tantalum implant inserted in a later procedure.[9]

In 1948 Lewin, Graham and Northcroft[10] evaluated the immediate and long term results of 130 tantalum cranioplasties performed on 128 patients in British military hospitals. All of their patients had been operated on at least two years previously. Of this group, 110 had completely uneventful surgical recoveries. Follow-up examinations were made two years or more after the operations in 121 of their patients; 94 of them were completely asymptomatic. The remaining 27 complained of slight to moderate pain, intracerebral complications, poor cosmetic results, or infection. These writers observed that the highest incidence of failure involved plates which covered regions that readily became infected such as the frontal sinuses, and further observed that plates covering the frontal area tended to drift downward unless they were carefully fitted and firmly attached to the surrounding bone.

In 1953 Kiehn and Grino[11] reported a series of ten tantalum cranioplasties

3. Pudenz, R. H. Repair of cranial defects with tantalum, an experimental study. J.A.M.A. 121:478 Feb. 13, 1943.

4. Craig, W. M. War wounds of peripheral nerves. U.S. Nav.M.Bull. 41:613 May 1943.

5. Horrax, G. Panel discussion, treatment of war injuries of skull and brain. Bull. Am.Coll.Surg. 27:127 April 1942. Discussion by Capt. F. R. Hook, M.C., U.S.Navy.

6. Gerry, R. G. Simplified technique for fabrication of tantalum skull plates. U.S. Nav.M.Bull. 46:1499 Oct. 1946.

7. Schork, C. J.; Kaires, A. K., and Clare, F. B. Tantalum in cranioplasty and oral surgery. J. Oral Surg. 6:31 Oct. 1948.

8. Lane, S., and Webster, J. E. Report of early results in tantalum cranioplasty. J. Neurosurg. 4:526 Nov. 1947.

9. Schultz, A. L. Personal communication.

10. Lewin, W.; Graham, M. P., and Northcroft, G. B. Tantalum in repair of traumatic skull defects. Brit.J. Surg. 36:26 July 1948.

11. Kiehn, C. L., and Grino, A. Iliac bone grafts replacing tantalum plates for gunshot wounds of skull. Am.J.Surg. 85:395 March 1953.

which had failed, and in which they had replaced the tantalum plates with iliac bone grafts. The reasons they listed for tantalum plate failure were infection, especially in the region of the frontal sinuses; pain, especially as the result of heat and cold; erosion of the plates through pedicle flaps or into the orbit; and unsatisfactory cosmetic results.

King[12] and Turner[13] have both reported the successful use of plate tantalum for the repair of traumatic defects of the orbital roof. In 1950, Callahan[14] described the use of an unperforated tantalum plate to reconstruct the orbital floor and rim of an anophthalmic patient on whom he operated in 1945. In this instance, the plate was inserted to support an ocular prosthesis. The patient was followed and still had a good result three years postoperatively. Newman[15] also made use of unperforated tantalum implants for the correction of a defect of the orbital rim in one patient and a deformed zygomatic arch in another. Apparently the implant was not used to correct diplopia in either instance.

In addition to the foregoing, tantalum has been used successfully by Bellinger[16] and others for the plating of complicated mandibular fractures. Thoma[17] has reported the use of tantalum gauze for the correction of microgenia, and Holland[18] has used a similar material for alveoloplasties. In 1953, Scharfe[19] reported the use of tantalum in the correction of laryngeal stenosis and stenosis of the ear canal.

■ *Perforated Tantalum Plates in Facial Reconstruction*

On the basis of the foregoing reports and our experience since 1945, the use of perforated plate tantalum has been considered feasible for certain types of facial reconstruction.

USE OF PLATE TANTALUM TO RESTORE CONTOUR OF ORBITAL FLOOR AND MALAR PROMINENCE—The management of the malunited fractured malar prominence with depression of the orbital floor is one of the most perplexing problems in the field of facial surgery. In such instances there usually are two serious deformities; diplopia due to depression of the orbital floor and loss of normal facial contour as the result of loss of the malar prominence. If diplopia is not present, the loss of the malar prominence can be corrected easily. However, long-standing diplopia which has not responded to exercise or revision of the extraocular muscles is extremely difficult to correct. This problem is made increasingly difficult when concurrent enophthalmus exists. Fortunately, some of these patients respond well to surgical exposure of the fracture lines and refracture of the malar prominence many months after injury. When successful refracture cannot be accomplished, however, some means must be found to reconstruct the orbital floor. In most instances this correction is attempted by in-

12. King, A. B. Traumatic encephaloceles of orbit. A.M.A. Arch.Ophth. 46:49 July 1951.

13. Turner, O. A. Repair of defects of skull, with special reference to periorbital structures and frontal sinuses. Arch. Surg. 53:312 Sept. 1946.

14. Callahan, A. Surgery of the eye: injuries. Springfield, Ill., Charles C Thomas, 1950, p. 195.

15. Newman, J. Use of tantalum for facial and cranial defects. Am. J. Surg. 73:499 April 1947.

16. Bellinger, D. H. Use of tantalum in maxillofacial and oral surgery; a preliminary report. J. Oral Surg. 5:108 April 1947.

17. Thoma, K. H. Genioplasty with tantalum gauze. Oral Surg., Oral Med. & Oral Path. 2:65 Jan. 1949.

18. Holland, D. J. Alveoplasty with tantalum mesh. J. Pros.Den. 3:354 May 1953.

19. Scharfe, E. E. Use of tantalum in otolaryngology. A.M.A.Arch. Otolaryng. 58:133 Aug. 1953.

Fig. 1—Specimen of tantalum orbitomalar plates showing multiple perforations, flanges for correction of deformities of orbital floor and malar prominence, and counter-sunk drill hole to accommodate a tantalum screw

serting slivers of bone or cartilage, subperiosteally, and elevating the globe gradually until its malposition has been corrected. This procedure leaves something to be desired, as it is difficult in this manner to reposition the eye sufficiently precisely, and if surgical repositioning is successful, postoperative resorption of the graft will alter the position of the eye. In this important respect, tantalum, or any nonabsorbable material, is superior to any physiologic type of graft.

In 1948, one of us (R.G.G.) made use of a perforated tantalum implant with an orbital surface to restore a deformed orbital floor and a facial surface to restore the depressed malar prominence (Fig. 1). The patient recovered uneventfully. After a few months he failed to return for follow-up visits.

Since that time the writers have inserted two similar tantalum implants, one in December 1953 and the other in July 1954. Both patients have been followed carefully, and neither has had any complaints.

TECHNIC OF CONSTRUCTING PLATE TANTALUM IMPLANTS—When plate tantalum implants to correct deformities of the orbital floor and malar prominence are constructed, the bony contours of both orbits are palpated and marked on the skin with indelible pencil, and the vertical and horizontal dimensions of the uninjured orbit are measured and recorded (Fig. 2). An alginate impression of the face is taken and poured in laboratory plaster. The penciled orbital rim markings will be transferred to the plaster model.

A skull is selected which has the same orbital dimensions as the patient's uninjured orbit. The anatomic undercuts are eliminated with wax in the skull orbit which corresponds to the patient's deformed orbit. This orbit is lubricated and a plaster impression taken of it. This impression is used as a template, and roent-

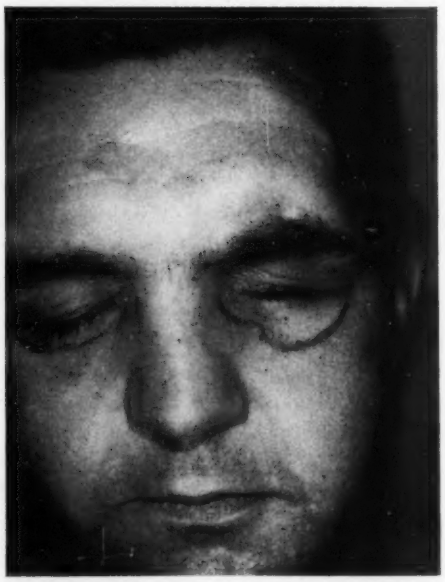

Fig. 2—Outline of deformed orbital floor marked with indelible pencil prior to making moulage of face (Case 1—orbitomalar operation)

genograms and palpated landmarks are used as guides when the orbital contour of the skull orbit is carved into the plaster model of the patient's face. The foregoing procedure is painstaking, but may be accomplished readily with the assistance of any conscientious dental intern or prosthetic technician. It will more closely approximate the patient's original orbital contour than when any less tedious method is used. The malar contour is then modeled in Plasticine to conform to that of the uninjured side. The outline of the implant is marked in indelible pencil around the molded Plasticine malar reconstruction and over that part of the orbital floor which is to be reconstructed (Fig. 3).

The implant area is then lubricated, and an artificial stone die poured. The die should extend at least one inch beyond the periphery of the plate in all directions, and should be at least two inches thick at its thinnest point. The die is keyed at three or four points on its periphery, and an artificial stone counter-die poured. A sheet of clear Plexiglas is flamed until it bends freely; it is then pressed between the die and the counter-die. The penciled outline on the die may be seen through the Plexiglas which is trimmed to conform to the penciled periphery. The Plexiglas is reflamed and pressed flat. The flattened Plexiglas plate will show the outline accurately, in a flat sheet which a tantalum plate must have in order to be pressed to the proper contour for the implant. This outline is scratched on a sheet of 0.0125 inch or 0.0150 inch plate tantalum, which may be trimmed appropriately.

A machine shop type of punch drill or press, if available, is used to drill as many holes as possible through the plate. The size of the holes is not too important, so any convenient drill may be used. Probably 2 to 3 mm. holes drilled 2 or 3 mm. apart will be suitable in most instances.

The perforated tantalum plate is then placed on the counter-die and "worked" with a round-end maple dowel and mallet so that it conforms roughly to the counter-die. At points where wrinkles appear, appropriate V-shaped wedges should be cut at right angles to the periphery. Final contour should be developed by pressing the plate between the die and the counter-die. At this point one of the drill holes in the malar area should be enlarged and counter-sunk to accommodate the tan-

Fig. 3—Plaster model of face showing penciled outline of uninjured orbit, and Plasticine reconstruction of deformed orbital floor and malar prominence (Case 2—orbitomalar operation)

talum retention screw which will be used during the insertion of the plate. The plate is then smoothed but not polished on a lathe, and the edges are tapered. At this point the plate may be placed on the original plaster model; it should conform exactly to the corrected orbital floor-malar deformity. The implant is then scrubbed thoroughly, wrapped and autoclaved (Fig. 1).

INSERTION OF PLATE TANTALUM IMPLANTS—The insertion of the plate is actually a much simpler procedure than its preparation. A skin incision is made just below the site of the inferior orbital border, in a fold. It should be just long enough to permit the insertion of the plate. The periosteum is exposed and split and elevated over the orbital floor and malar regions enough to permit the implant to be slipped into position.

The implant should contact bone over its entire periphery, but if it does not, the periphery may be adjusted at this time. The level of the inferior orbital rim and the pupillary level are then compared carefully with those of the uninjured orbit. This comparison can be made more accurately if the patient is placed in a sitting position. If additional adjustment of the orbital flange is necessary, the ophthalmologist may accomplish it with a pair of thin-nosed pliers without removing the implant. When satisfactory position of the globe has been established, the medial edge of the implant should be wired to the uninjured bone of the orbital rim with tantalum wire (Fig. 4, left). The lateral margin of the plate is fastened to the malar prominence with a tantalum screw placed in the hole which has already been prepared for its reception (Fig. 4, right). The periosteum is then closed over the implant, if possible, and the skin and subcutaneous tissues are closed without tension, even though further undermining may be necessary.

■ *Report of Cases*

CASE 1—A 29 year old man had been admitted to another hospital on August 1, 1953, a few hours after having been in an automobile accident. At that time the patient was unconscious; he had suffered extensive comminuted fractures of the frontal bone, and multiple fractures of the occiput, nasal bones, maxilla, left malar prominence, left zygomatic arch and mandible.

No attempt had been made to reduce any of the fractures at the hospital to which the patient had been originally admitted. He was subsequently transferred to the U. S. Naval Hospital, St. Albans, N. Y., where his maxillary and mandibular fractures, that had partially repaired in malposition, were separated and anatomically reduced. The position of the left malar prominence and orbital floor could not be substantially improved (Fig. 2).

Re-evaluation of the patient on November 27, 1953, indicated that the following conditions were still present: depression of skull over the left frontal sinus; depression of the left malar bone and orbital floor; gross depression and enophthalmos of the left eye with vision of 20/200; an upper medial quadrantic field defect of the left eye, probably due to optic nerve damage at the optic foramen; and severe limitation of motion of the left eyeball.

On December 2, a tantalum plate was inserted over the left frontal defect to fill out this depression, and a shaped tantalum plate with malar and orbital flanges was inserted to correct the malar depression and to raise the left eye (Fig. 4, left).

The patient recovered uneventfully from surgery. He was re-evaluated on January 6, 1954. The insertion of the tantalum plates had produced a good cosmetic result, had materially reduced the enophthalmos, and had raised the left eye to the same level as the right eye. He retained the upper, inner quadrantic field defect of the left eye, but vision of this eye had improved to 20/50 corrected to 20/40. Funduscopy showed partial optic atrophy of the left eye. There was still some limitation of motion of the left eye, but this had decreased significantly over that which had existed preoperatively (Fig. 5).

The patient has been returned to full duty and has been contacted, through his Station Medical Officer, twice yearly since his discharge from the hospital. He was last contacted in December 1955, and he reported that he was completely asymptomatic except for those residual defects of the left eye which had been present when he was discharged from the hospital.

CASE 2—A 20 year old man had been in an automobile accident on December 31, 1953. He had been admitted to another hospital after the accident and had

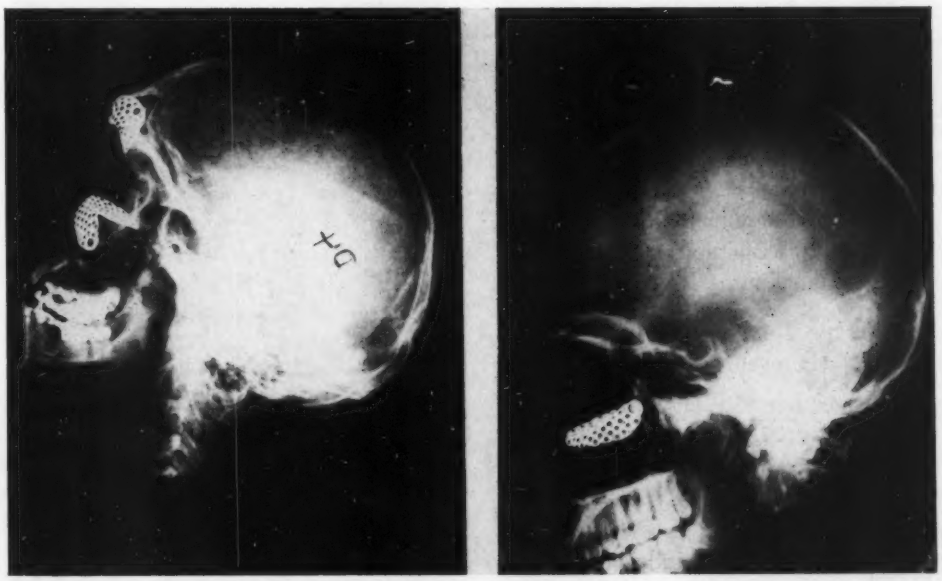

Fig. 4—Left: Lateral roentgenogram of patient (Fig. 2) showing implant wired in position. Right: Lateral roentgenogram of patient (Fig. 3) showing tantalum implant with tantalum screw in malar prominence

been unconscious for seven days. Blindness of the left eye had been evident since the day he had recovered consciousness. An attempt at plastic repair of the left maxilla had been made on January 11, 1954.

He was admitted to the U. S. Naval Hospital, St. Albans, N. Y., on May 5. At that time he had complete loss of vision in the left eye, and malunited fractures of the left supraorbital ridge and the left malar and orbital floor which were depressed. On May 13 the supraorbital deformity was corrected by the subperiosteal insertion of a piece of preserved cartilage about 2 by 4 by 8 cm. A tantalum plate with malar and orbital flanges was then prepared to correct the malar deformity and to elevate the left eye to the level of the right (Fig. 3, 4, right). This was inserted on July 22. The patient had an uneventful recovery, and the operation resulted in normal facies with normal position of the left eye.

The patient was separated from the Naval Service because of unilateral blindness on September 14. Since that time he has been contacted twice by letter. He reports that he has no noticeable deformity, and that he is completely asymptomatic except for the blindness in the left eye.

■ *Plate Tantalum for Correction of Microgenia and Micrognathia*

In addition to using perforated tantalum implants for the correction of acquired deformities of the malar and orbital floor, one of us (R.G.G.) has used similar plates for the correction of microgenia and micrognathia. It has already been noted that Thoma[17] reported the use of tantalum mesh for the treatment of a similar deformity. Plate tantalum offers certain advantages over mesh in that it can be carefully and accurately contoured preoperatively, thus materially reducing the length of the procedure and assuring a good cosmetic result if it is possible to obtain one. It is also easier to remove tantalum plate than tantalum mesh, if removal of the implant should become necessary. Tantalum plate also has the following advantages over bone and cartilage:

1. There is no resorption of the implant and consequently no change in the patient's postoperative appearance.

2. There is no collateral procedure at a donor site, as, for example, in the iliac crest. Hospitalization is not required for

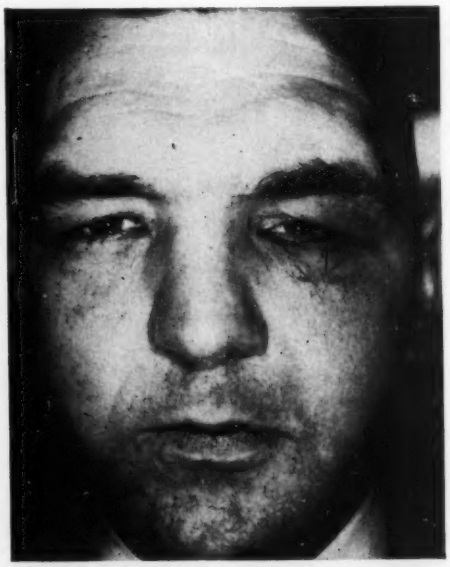

Fig. 5—Early postoperative view of Case 1 (orbitomalar operation) showing similar right and left malar contour and pupillary level of eyes. Healing infraorbital incision is barely noticeable

the insertion of a tantalum plate in genioplasty.

To date, tantalum genioplasty has been performed in two patients with highly gratifying results. Subsequently Stanley L. Lane[20] has achieved equally satisfactory results with the same operation in two patients.

FABRICATION OF TANTALUM GENIOPLASTIC IMPLANT—The technic for fabrication of a tantalum genioplastic implant is very similar to that which has already been described for the preparation of tantalum implants for malar bone-orbital floor deformities. An impression of the face is taken, and a model is poured in plaster or artificial stone. If concurrent overjet of the maxillary incisors exists, and this deformity is to be corrected, this procedure should be completed, if feasible, before the facial model is made. The receding chin is corrected in Plasticine on the model, and an artificial stone die is poured. An artificial stone counter-die is then made from the die. The outline of the tantalum plate is established by the use of a molded Plexiglas plate as described earlier, with one exception. Two pointed prongs, about 2 to 4 cm. apart, are cut out from what will be the superior margin of the implant. These should be 2 to 3 mm. across the base, and 3 to 4 mm. long. These prongs will be used at the time of insertion to nail the implant into the bony cortex. Holes are then drilled in the plate as described earlier, and it is then contoured in the same manner.

INSERTION OF PLATE TANTALUM IMPLANT FOR GENIOPLASTY—To insert the plate tantalum implant for genioplasty, an incision is made through the mucous membrane of the lower lip, about halfway up its oral surface. The incision should be parallel to the labial sulcus and need be only slightly more than one half the length of the implant. The mucous membrane is undermined and elevated inferiorly and posteriorly until the mandibular periosteum is reached, over the mental protuberance. The smallest feasible incision is used when the periosteum is split, so that the periosteum will help anchor the implant. A subperiosteal pocket, just large enough to accommodate the implant, should be elevated. This pocket should extend just to the inferior border of the mandible. The implant may be tried at this point and necessary adjustments made, but as a precautionary measure, a wire ligature should be passed through a pair of suitably placed holes so that if removal is required, the wire can be used to provide traction. If the implant is entirely satisfactory, its superior margin should be pulled away from the mandible to permit the two prongs to be bent at right angles to the mandibular cortex. The wire ligature may then be withdrawn, and the implant pushed down 2 or 3 mm. below its correct position. Holes are then made in the cortex with a sharp instrument, so that the prongs will engage the holes when the implant slides upward into its correct position. At this point the implant should be anchored firmly in correct position (Fig. 6).

Throughout the procedure hemorrhage should be controlled carefully so that there will be no postoperative pooling of blood in the dead space between the

20. Lane, S. L. Personal communication.

mandible and the implant. The periosteum is closed, if possible, and the mucous flap returned and closed without tension. A strip of elastic adhesive tape is placed over the skin, between the top of the implant and the lower lip, to immobilize the soft tissues.

■ *Report of Cases*

CASE 1—A 23 year old woman came for treatment in March 1952. Her principal complaint was dissatisfaction with her own facial appearance. Examination of the patient indicated that she had considerable protrusion of her maxillary incisors, nasal obstruction with deformity, and severe recession of the chin (Fig. 7, left). Her maxillary incisors were extracted with suitable alveolectomy, and an immediate partial denture was inserted to provide a normal incisal relationship.

In April 1953 a rhinoplasty with submucous resection was performed and, at the conclusion of this procedure, a homologous cancellous bone graft was inserted through a submental incision to correct the microgenia. The patient's postoperative appearance was excellent, but it became evident during the next few months that the bone graft was being absorbed.

In January 1954, a prepared perforated tantalum implant was inserted through the intraoral approach. This increased the prominence of the patient's chin by about 12 mm. (Fig. 6, right). The patient's postoperative course was uneventful and her improved facial contour has been maintained. She has been

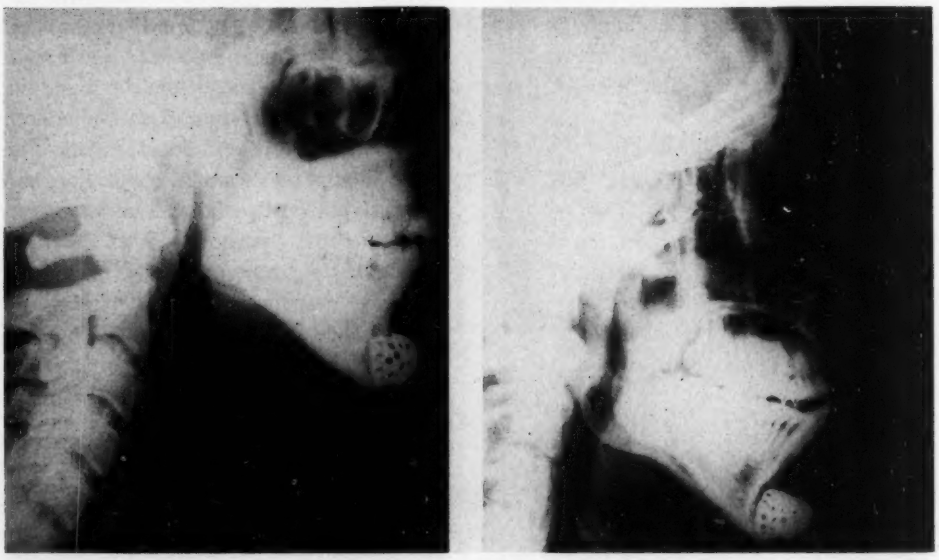

Fig. 6—Left: Lateral roentgenogram showing tantalum implant in genioplasty patient (Case 1). Corrected overjet of maxillary incisors is also demonstrated. Right: Lateral roentgenogram showing tantalum implant in genioplasty patient (Case 2)

Fig. 7—Left: Preoperative profile view of genioplasty patient (Case 1) showing typical facial deformities. Right: Postoperative profile view of same patient after rhinoplasty, correction of incisal overjet, and genioplasty

observed periodically since genioplasty, and she reports that she has been entirely symptom-free. Not only is she completely pleased with her facial improvement, but she reports that she has been able to obtain part-time employment as a portrait model (Fig. 7).

CASE 2—A 21 year old man had been admitted to the hospital with a diagnosis of psoriasis. At the conclusion of his dermatologic therapy he also expressed dissatisfaction with his facial appearance. Examination demonstrated the typical facies of many microgenic patients; that is, deformed nose with obstruction, extreme overjet of the maxillary incisors, and a "weak" chin (Fig. 8, left).

On January 18, 1955, the four maxillary incisors were extracted and alveolectomy was performed. The alveolar process to be sacrificed was resected en bloc, and an immediate partial denture which provided a satisfactory incisal relationship was inserted. At the conclusion of this procedure, a prepared plate tantalum implant was inserted through the intraoral approach to correct the patient's microgenia (Fig. 6, right). The resected block of maxillary alveolar process was placed under the implant to eliminate the dead space.

The patient's postoperative course was uneventful except for slight hemorrhagic drainage from the incision which started on the seventh postoperative day. The drainage ceased after 11 days following daily irrigation with one million units of aqueous penicillin for three days.

On March 1, 1955, rhinoplasty with submucous resection was performed. The patient was followed for six months postoperatively. Apart from the short period of drainage described, he has been completely asymptomatic and entirely satisfied with his facial appearance (Fig. 8).

■ *Discussion*

Perforated plate tantalum is a highly satisfactory implant material for those types of facial reconstruction which require such careful consideration of the revision of the facial skeleton that it may be desirable to use an implant which can be constructed in advance of surgery. Tantalum satisfies this requirement in a more satisfactory manner than any other material. In some instances, as in reconstruction of the orbital floor for the correction of diplopia, it may be preferable to autogenous bone, as no postoperative changes in the position of the globe will occur. Tantalum may be used safely for the types of facial reconstruction described earlier provided the following criteria are satisfied:

1. Tantalum plates should not be placed in infected sites. In traumatic injuries in which the maxillary sinuses may have been involved, surgery should be

Fig. 8—Left: Preoperative profile view of genioplasty patient (Case 2) showing typical facial deformities. Right: Postoperative view of same patient after rhinoplasty, correction of incisal overjet and genioplasty

postponed until all fistulas from the sinuses have closed and all sinusitis has resolved.

2. The plates should be contoured carefully to provide anatomic contour and should fit the bony surfaces snugly over the entire periphery of the plate. If the covering tissues are adequate, excellent cosmetic results can always be obtained if the plate has been contoured properly.

3. The plate should be fastened in place firmly by means of tantalum screws, prongs or wires which actually engage bone, so there will be no danger of the plate slipping and eroding through the covering tissues.

4. The plate should be covered with as thick a soft tissue flap as possible to assure adequate vascularity of the covering tissues and to provide as much insulation as possible to the plate so that it will not conduct thermal stimuli from the body surface. If there is extensive scarring of the covering tissues, the indicated plastic procedures should be completed and allowed to heal before the plate is inserted.

5. The plate should be perforated with as many holes as possible, consistent with adequate strength, to permit passage of fluid during the early postoperative period and to provide the best possible trellis for fibrosis after this change occurs. Multiple perforation also permits more accurate contouring of the plate itself and, by reducing the metallic mass, assures a much higher proportion of insulating tissues and consequently a much lowered incidence of conduction of thermal stimuli from the body surface.

■ *Conclusions*

Perforated plate tantalum implants are useful in the reconstruction of the orbital floor for the correction of diplopia and the re-establishment of the malar prominence and for the correction of microgenia because they can be contoured accurately and carefully in advance of surgery, and because no postoperative changes in contour occur. The latter characteristic is of particular importance in the surgical correction of diplopia resulting from deformity of the orbital floor.

Properly contoured tantalum plate implants may be inserted safely in sites which are free from infection, provided the implant fits the bony surface over its entire periphery, is properly fastened to the underlying bone, is covered by an adequate flap of healthy soft tissue, and is suitably perforated.

■ Transmission of Ideas

From the point of view of the history of science, transmission is as essential as discovery. If the results of Ptolemy's investigations had been hidden instead of published, or if they had been lost in transit, they would be almost as if they had never been.—*George Sarton, Introduction to the History of Science, vol. 1.*

Condylectomy for the persistently painful temporomandibular joint

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The treatment of the painful temporomandibular joint has been an interesting and persistently difficult problem for many years.[1-4] Various types of surgical intervention have been advocated for conditions that have resisted conservative therapy, but in general none has produced permanent relief and good function. Meniscectomy has been advocated by several surgeons,[4-7] but unfortunately, despite usual early relief, many patients develop recurrence of symptoms at a later date. Condylectomy generally has been in disfavor because of the probability of postoperative jaw deviation, general disruption of the jaw function, and the possibility of damage to the facial nerve.

We have tried various surgical procedures in an attempt to provide permanent relief from symptoms and still retain normal function. Meniscectomy was utilized for a period, but was discarded because of a rather high rate of persistent or recurrent pain. Condylectomy combined with meniscectomy was then tried, and although it produced relief, it was not considered desirable because of rather noticeable postoperative jaw deviation. Preserved bovine cartilage was used in a few patients to replace the condylar section which was excised. Results were again unpredictable, however, because of the inability of the operator to anchor and control the cartilage fragment adequately.

In 20 patients, a high condylectomy has been performed, leaving the condylar neck, meniscus, ligamentous attachments and surrounding capsule intact and relatively undisturbed (Fig. 1). This procedure has produced universally excellent results, since occlusion remains comparatively unchanged (Fig. 2) and pain is completely eliminated. There have been no indications of facial nerve damage, and cosmetic results have been uniformly good (Fig. 3). In reviewing these patients critically, it was found that in 50 per cent, postoperative occlusion was normal and no measurable deviation occurred when the mandible was in the extreme open

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2. Costen, J. B. Syndrome of ear and sinus symptoms dependent upon disturbed function of temporomandibular joint. Ann.Otol.,Rhino. & Laryng 43:1 March 1934.

3. Bauer, W. H. Osteo-arthritis deformans of temporomandibular joint. Am.J.Path. 17:129 Jan. 1941.

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5. Dingman, R. O., and Moorman, W. C. Meniscectomy in treatment of lesions of temporomandibular joint. J. Oral Surg. 9:214 July 1951.

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position. In the remaining 50 per cent, slight deviation up to 3 or 4 mm. occurred on wide opening, and in three there was sufficient change in the closed position so that some slight occlusal adjustment was made. In no instance was there considerable or undesirable deviation. When some degree of deviation was present, it was so insignificant that the patient was not cognizant of it.

Roentgenographically, the joint which has been operated on shows early rounding off of the severed stump. In a few instances some regeneration has seemed to be present (Fig. 4). Clinically, it appears that this procedure allows regeneration of the joint to the point that function returns to a normal state. Apparently the presence of the meniscus prevents formation of fibrous adhesions between the glenoid fossa and the resected stump; thereby, postoperative deviation and restriction of motion are prevented. Resection of the condyle removes any possible pressure or traction on the meniscus or on the area of nerve supply posterior to the meniscus, and immediate relief from pain is provided.

In his recent report, Sicher[8] provided a new explanation regarding the innervation of the temporomandibular joint which gives an anatomic reason for the success in obtaining universal relief of pain after the high mandibular condylectomy. Sicher states:

> Although the medial, anterior and lateral edges of the articulating disk are directly attached to and fused with the capsule, the posterior border of the disk is bound to the capsule by loose connective tissue, that in sagittal section presents a triangular shape (Fig. 5). In contrast to the pressure bearing and thus avascular and nerveless disk, this loose connective tissue is rich in blood vessels, nerves and nerve endings, the latter supplied by branches of the auriculotemporal nerve.

He also points out that the loose connective tissue between the disk and the posterior portion of the capsule is the most important point of low resistance, and therefore is the most susceptible region for mechanical damage to the temporomandibular articulation. Sicher's contribution to this field of study is of paramount importance, since it opens the way to a better understanding of joint pain and dysfunction. It also indicates the need for decreasing pressure on the disk and on the nerve supply posterior to it, especially in patients that do not respond to conservative therapy. Excision of the condyle, therefore, can be expected to relieve any pressure or traction on this region of nerve supply.

In the study, only patients who had had persistent pain in spite of extensive long range conservative therapy were included (see table). Many also had had distinct limitation of motion. The patients have been divided into four main classifications:

1. Those whose joints have varying degrees of fibrosis with limitation dating back to a single act of trauma. The tension factor usually has been superimposed.

2. Those with chronic subluxation, with pain and audible snapping during excursion of the condyle.

3. Those with arthritic changes resulting from degenerative joint disease, including narrowing of the joint space, osteophyte production, erosion of the articular surface and limitation of motion.

8. Sicher, Harry. Structure and functional basis for disorders of temporomandibular joint. J. Oral Surg. 13:275 Oct. 1955.

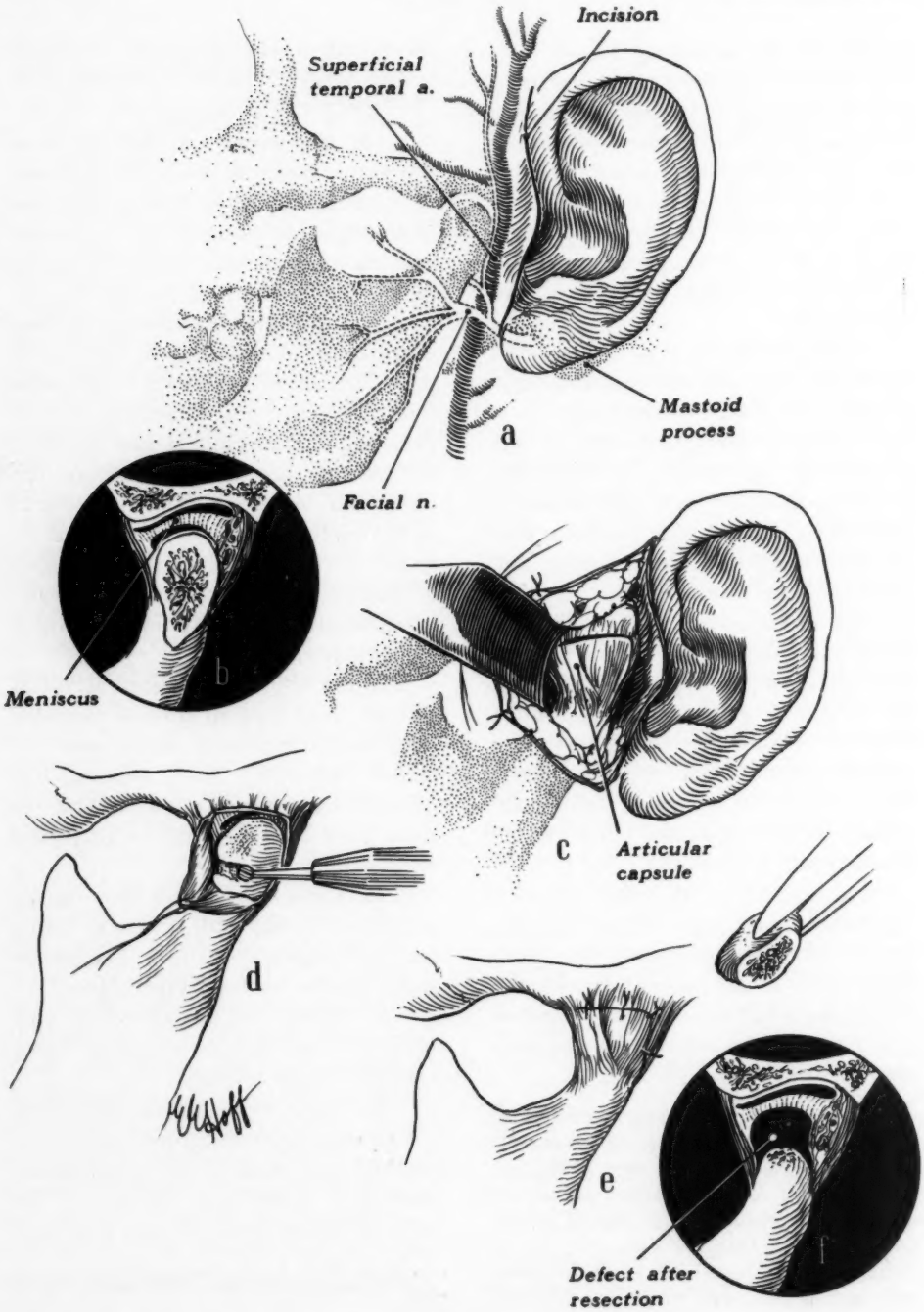
Incision
Superficial temporal a.
Mastoid process
a
Facial n.
Meniscus
b
c
Articular capsule
d
e
Defect after resection
f

Fig. 1—Operative procedure: (a) Incision showing relation to facial nerve, temporal vessels and ear; (b) sagittal section of temporomandibular joint showing meniscus and nerve-bearing loose connective tissue immediately posterior to it; (c) incision in capsule to expose condyle; (d) drill cut at base of condyle made with tungsten carbide drill in dental handpiece; (e) capsule sutured; (f) postoperative sagittal section of temporomandibular joint with rounding off of condylar neck and preservation of meniscus. Illustrates elimination of pressure on meniscus and nerve-bearing tissue

4. Those with a tension syndrome—spasm of the mandibular musculature with limitation of opening and deviation from the midline, with no roentgenographic evidence of joint alteration.

All patients had had extensive conservative therapy that ranged from 3 to 60 months. Therapy included rest to the joint (by interdental ligation in five instances), soft diet, occlusal equilibration, intermittent heat therapy, full mouth reconstruction (in two cases), barbiturates and muscle relaxants for reduction of muscle tension and ultimately intra-articular hydrocortisone injection.[9] In all instances it eventually became obvious that conservative treatment was inadequate. In several instances patients experienced temporary relief after intra-articular hydrocortisone injection, only to have recurrence of pain in two to four weeks. In those with limited mandibular function, exercise only served to increase pain in the joint region. All were subsequently given complete relief by high mandibular condylectomy in which the meniscus, the condylar neck and surrounding attachments were left intact. Postoperatively all mandibular movements and masticating function rapidly returned to normal.

■ *Operative Procedure*

All hair is carefully shaved off on the operative side for a distance of one inch above and behind the ear. After the usual skin preparation is done, the tissues anterior to the ear are generously infiltrated with an epinephrine-containing local anesthetic solution to aid in anesthesia and hemostasis. The incision is made immediately anterior to the auricular cartilage and extends from the anterior margin of the helix, inferiorly behind the tragus and then down to the attachment of the lobe (Fig. 1, a).

The skin anterior to the incision is then undermined for approximately 1.5 cm. along its entire length, and is sutured forward to aid in access and exposure. By careful sharp dissection, the soft tissues are separated from the cartilage until the zygomatic arch is reached. This is an important landmark and must always be visualized. Care should be exercised to refrain from cutting into the cartilage, but usually the dissection can be accomplished rapidly. It is imperative to maintain the plane of dissection in contact with the cartilage to insure preservation of the facial nerve and temporal vessels which richly supply the area.

As the dissection proceeds and the wound is deepened, the soft tissues anterior to the ear are retracted forward by the assistant. When the zygomatic arch

9. Henny, Fred. Intra-articular injection of hydrocortisone into temporomandibular joint. J. Oral Surg. 12:314 Oct. 1954.

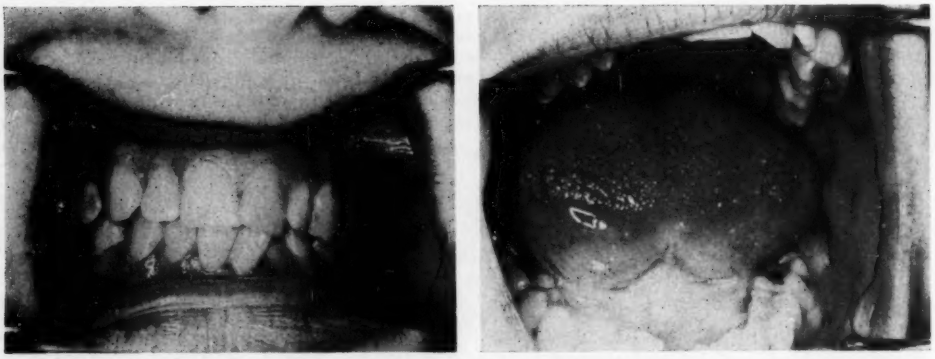

Fig. 2—Left: Postoperative condylectomy with teeth in occlusion. Note retention of normal midline despite lack of postoperative immobilization. Right: Same patient; mandible in extreme open position with ample painless excursion and no deviation

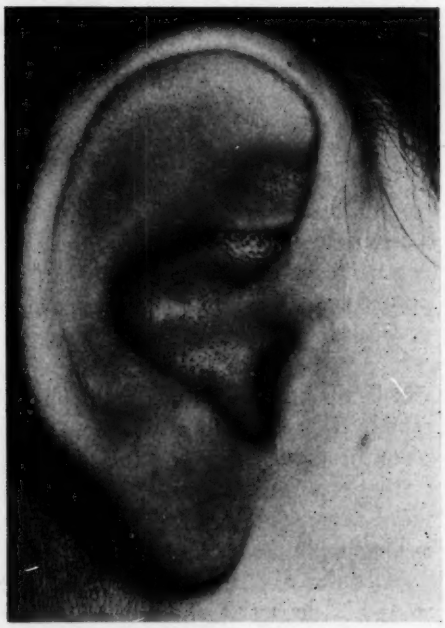

Fig. 3—Postoperative incision line following complete healing

landmark is reached, the wound is deepened an additional 0.5 cm. before the dissection is extended anteriorly. The soft tissues are than dissected along the zygomatic arch where a small vein is consistently located. It is transected and tied. The capsule of the joint is exposed by anterior blunt dissection and is opened through an L shaped incision (Fig. 1, c). The condyle is exposed; care must be taken to avoid the meniscus, but otherwise the entire head of the condyloid process and a portion of the neck are exposed to aid in orientation when the level of resection is planned.

Resection should be done at the base of the condyle and should not be carried down on to the neck since there is no benefit in so doing and it may contribute to postoperative deviation (Fig. 1, d). Only an amount sufficient to remove pressure on the meniscus or the nerve-bearing tissue posterior to it is required (Fig. 1, b, f). An angular cut can be used, if desired, to decrease contact posteriorly and yet retain as much height anteriorly as possible.

The actual sectioning is done with a small round tungsten carbide dental drill under water irrigation. The cut is extended medially until the head of the condyloid process is almost severed. The osteotomy can then be completed with a light tap of a chisel. This is done to eliminate the possibility of vessel damage medial to the condyle. The resected condyle is then grasped with a forceps and is delivered by stripping off any of its remaining attachments with a periosteal elevator. Some oozing may ensue, but this is easily controlled with a Gelfoam pack.

Fig. 4—Postoperative roentgenogram showing jaw in open position. Note normal anterior gliding action of ramus and resected stump

The resected stump is smoothed with a bone file, Gelfoam is placed in the defect, the capsule is sutured (Fig. 1, e), and the wound is closed in the usual fashion. A pressure dressing of generous size is applied over the area of operation and is left in place for three days.

Since early function is desirable, the patient should be encouraged to use the jaw as soon as possible. A soft diet usually can be started on the second postoperative day, and this can be increased in consistency as healing progresses. Because of spilling of blood from the incision into the external ear canal, it is usually wise to irrigate and cleanse the canal after completion of the procedure or at the time of the removal of the pressure dressing. The

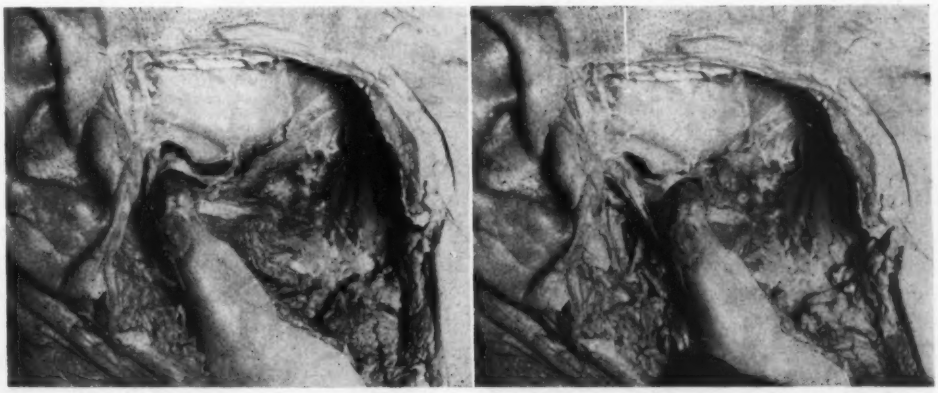

Fig. 5—Area of nerve-bearing loose connective tissue posterior to meniscus (from Sicher[3])

Table

Patients in whom high mandibular condylectomy was performed for relief of persistent pain

Classification	Age & sex of patient	Time from original trauma to time of operation	Period of conservative therapy*	Time since condylectomy	Post-operative occlusion	Relief of pain
Varying degrees of fibrous and bony ankylosis with limitation of motion and pain after single act of trauma	39 fe.	5 mo. Fracture	3 mo.	6 mo.	†	Complete
	43 ma.	6 mo. Fracture	4 mo.	7 mo.	Normal	Complete
	57 ma.	7 mo. Blow	5 mo.	24 mo.	†	Complete
	43 ma.	7 mo. Fracture	4 mo.	36 mo.	Normal	Complete
	15 ma.	8 mo. Fracture	5 mo.	24 mo.	†	Complete
	26 fe.	13 mo. Fracture	11 mo.	19 mo.	Normal	Complete
	31 fe.	5 yrs. Fracture	4 mo.	24 mo.	Normal	Complete
	26 ma.	17 yrs. Blow	2 mo.	9 mo.	Normal	Complete
	45 fe.	12 mo. Blow	4 mo.	14 mo.	Normal	Complete
Chronic subluxation with pain and snapping noise	36 fe.	No trauma	4 mo.	30 mo.	Normal	Complete
	27 fe.	No trauma	60 mo.	30 mo.	†	Complete
	30 fe.	No trauma	24 mo.	24 mo.	†	Complete
Arthritic changes with narrowing of joint space and limitation of motion	52 fe.	No trauma	7 mo.	36 mo.	†	Complete
	51 fe.	No trauma	9 mo.	15 mo.	Normal	Complete
	37 fe.	No trauma	6 mo.	36 mo.	Normal	Complete
	32 fe.	No trauma	3 mo.	18 mo.	†	Complete
	30 fe.	No trauma	4 mo.	5 mo.	Normal	Complete
	38 fe.	No trauma	5 mo.	12 mo.	†	Complete
Tension syndrome—muscle spasm with deviation from centric	52 fe.	No trauma	6 mo.	14 mo.	†	Complete
	28 fe.	No trauma	12 mo.	36 mo.	Normal	Complete

*Conservative therapy consisted of immobilization of joint, soft diet, heat, occlusal equilibration, ammobarbital sodium for tension relief and hydrocortisone injections.

†Postoperative occlusion: normal centric position with slight deviation to affected side on opening.

patient otherwise may complain of decreased hearing and tinnitus in the ear due to its occlusion with clotted blood.

Summary

An operative procedure for surgical relief of persistent temporomandibular joint pain has been developed. Twenty patients who had failed to respond to conservative therapy were subjected to high mandibular condylectomy with preservation of the meniscus, condylar neck and ligamentous attachments. All these patients have had complete and lasting

relief from pain, and function has returned to normal rapidly with no evidence of facial nerve damage. Very slight deviation of the mandible in its extreme open position was observed in 50 per cent of the patients, but was too insignificant to be of clinical importance. Operative interference is indicated only when comprehensive conservative therapy has failed to eliminate joint pain.

A modified metal urethral sound: a new instrument in reduction of zygomatic fractures

■ *Raymond E. Boudreaux, D.D.S., U. S. Army*

The zygoma is important anatomically as the structure that helps to form the lateral and inferior wall of the orbit and the natural prominence of the cheek. It is situated primarily in the lateral aspect of the facial skull and is in a vulnerable position with regard to trauma about the face. When depressed fractures of the zygoma are seen soon after they occur, flatness or depression of the area is apparent. Subsequent edema, however, tends to mask the cosmetic defect—hence, the consistency with which the diagnosis of the fracture is missed. Fractures of the zygoma are usually not difficult to correct but unfortunately, in many instances, they are made more complicated and at times impossible to reduce by delay in treatment due to lack of a correct diagnosis at an early date. In instances with minimal or no displacement, however, an operative procedure usually is not required. Clinical and roentgenographic observations are the important factors in determining the indications for operation.

Suraci[1] feels that the clinical observations are definite and of more importance than roentgenograms. This conclusion is based on his observation of numerous roentgenographic reports of little or no displacement in instances in which the zygoma was greatly displaced.

It is conceded generally that accurate roentgenographic interpretation of extent of displacement, position (rotation) of fragments and in some instances of the fracture itself, is difficult. Some of this difficulty can be attributed to superimposi-

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1. Suraci, A. J. Method of reduction of the zygoma. Am. J. Surg. 88:843 Dec. 1954.

tion or faulty technic or both in making the roentgenograms. Accuracy in roentgenographic interpretation can be aided considerably by utilizing three views of the facial area: (1) stereoscopic Waters' views of the facial bones; (2) zygomatic arch views and (3) an exaggerated Townes' exposure.

Any attempted classification of these fractures would serve no useful purpose. The characteristic displacement and comminution factors are governed by the type, direction and amount of applied force. In those cases that are limited to arch injury, in most instances, the zygomatic process of the temporal bone also is involved.[2]

The diagnosis should be based on the history and on clinical and roentgenographic observations. Outlined are the physical findings that may be present:

1. Edema.
2. Crepitation.
3. Palpable dehiscence or break in continuity or alignment of the infraorbital ridge, zygomaticofrontal suture line, zygomatic arch or (intraorally) of the inferior aspect of the zygomaticomaxillary suture line or zygomatic process of the maxilla.
4. Depression noted by bilateral digital comparison.
5. Hypesthesia of the infraorbital region as a result of injury or impingement of the infraorbital nerve.
6. Diplopia due to depression or displacement of the orbital floor and rim with relaxation of the extraocular muscles.
7. Difficulty in opening the mouth its normal distance or deviation of the jaw to the affected side because of contact of the depressed zygomatic arch by the coronoid process of the mandible. The coronoid process is also fractured occasionally.
8. Subconjunctival hemorrhage.
9. The pupil level may be lower on the affected side.
10. Unilateral nasal hemorrhage resulting from laceration of the antral membrane.
11. Emphysema, in patients with fracture of the antral wall, may occur after sneezing or blowing of the nose.[2]

Some of the methods that have been advocated for the reduction of the zygoma and arch are as follows:

1. Passing of a wire around the zygomatic arch and applying traction in the direction required for reduction.
2. Grasping of the fragment with a towel clip and manipulating it into its correct position.
3. Insertion of a corkscrew type appliance through an external incision with subsequent manipulation of the fragments.
4. Insertion of a screweye through an external incision, with manipulation and elastic traction.
5. Intraoral approach through an incision in the mucobuccal fold and insertion of a blunt instrument under the zygoma to elevate depressed fragments.
6. Making of an incision in the hairline in the preauricular area and passing of an instrument under the zygoma.
7. Introduction of an instrument into the antrum and elevation of the fragments from that direction.[3] An antral pack

2. National Research Council. Committee on surgery. Manual of standard practice of plastic and maxillofacial surgery. Philadelphia, W. B. Saunders Co., 1942, p. 72-75.

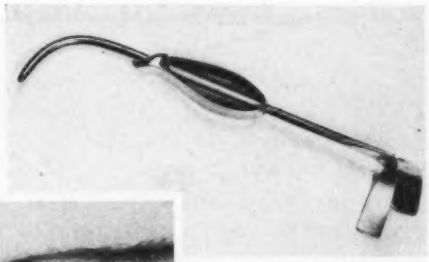
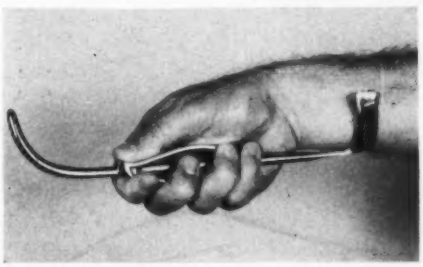


Fig. 1—Right: Modified urethral sound with hand grip and armrest



Left: Modified urethral sound grasped in position for use

is usually required to hold the fragments in position.

The metal urethral sound has been one of the instruments used for reductions of zygomatic fractures for years, both in the intraoral buccal approach and the sinus approach.[3]

The modified urethral sound (Fig. 1, right) was developed as a result of working with the plain sound and recognizing its inherent deficiencies when used for reductions of zygomatic fractures. These deficiencies were lack of stability; difficulty in exerting adequate pull force because of the problem involved in trying to grip a smooth and small metal rod tightly enough to prevent its slipping; the need to prevent lateral movement of the sound, in the region of the operator's wrist, to assure a maximum direct pull force. This latter point can best be explained by the fact that a greater amount of pull can be exerted on a rod fixed in its long axis to the arm, than a rod grasped by the hand with the arm at a varying angle (Fig. 1, left).

The welded attachments were designed to afford the means of overcoming these faults. The elliptical shaped addition near the point furnishes the hand grip, with a concavity at the upper side for a thumb rest. The U-shaped band welded to the base, provides a position for the wrist and fixes the arm in the long axis of the instrument, preventing lateral movement of the shaft and aiding stability.

The sound, because of its shape, is particularly well suited for reduction of zygomatic fractures. The author has used this modified sound since 1948, when the attachments were designed, on a series of 88 patients, and feels that it has distinct merit as an addition to the existing armamentarium. There are instances when other methods are indicated as the

3. Thoma, K. H. Oral surgery, vol. 1. St. Louis, C. V. Mosby Co., 1952, p. 473-475.

primary means of reduction, and in some of these instances the sound can be used as an adjunct. In fractures that are well fixed, that is, 12 to 21 days old, and that require open reduction, the sound can be used then to elevate and realign the depressed fragments. In those instances in which only the infraorbital ridge, floor of the orbit and antral wall are involved, the use of this instrument is precluded and the antral approach or the towel clip method or both usually are required.

In certain instances it may be necessary to use some means of stabilizing the reduced fracture. This need arises when the fragments do not retain their post-reduction alignment and tend to return to their former depressed position.

The use of a wire passed around the zygoma, and a plaster headcap with a metal extension for elastic traction, usually will suffice.[3] An antral pack also may be used for this purpose. Direct wiring must be resorted to occasionally when a satisfactory alignment cannot be achieved otherwise. This is particularly true in instances of transverse middle facial fractures.

■ *Operative Technic*

The patient is prepared in the usual manner. An intraoral incision approximately 2 cm. in length is made in the mucobuccal fold posterior to the zygomatic process of the maxilla. The buccinator muscle is separated, and the point of the sound is inserted into the infratemporal fossa.

The curved portion of the sound is brought into contact with the medial surface of the zygoma, and the blunt rounded point is extended to a point just beneath the skin superior to the body and arch of the fractured bone. This eliminates the possibility of the instrument's slipping forward and injuring the soft tissues when anterior force is applied. With the instrument fixed in the hand and to the wrist, pull force is exerted, while the opposite hand exerts counterpressure against the skull and digital palpation of the areas being reduced. The zygomatic arch is amenable to reduction as well when the point of the sound is angled posteriorly until it rests under the depressed fragments, then elevated by pull force, and realigned against digital pressure externally. Two or three sutures in the intraoral wound usually are sufficient for closure.

The following typical cases are reported.

CASE 1—An airman was admitted to Brooke Army Hospital, San Antonio, Texas, on December 15, 1948, after an injury incurred when he was struck by a motor scooter while crossing the street. He struck the left side of his face when he fell. There was a depression of the left malar area, which on palpation elicited no crepitation. Lack of symmetry of the left infraorbital ridge and hypesthesia of the infraorbital region extending to the side of the nose and the left upper lip was noted. No diplopia was present. Roentgenograms confirmed the clinical impression of fracture as shown in Figure 2, left.

The patient was operated on on December 21 under general anesthesia with the elevation and reduction accomplished with the modified urethral sound through an intraoral incision (Fig. 2, right).

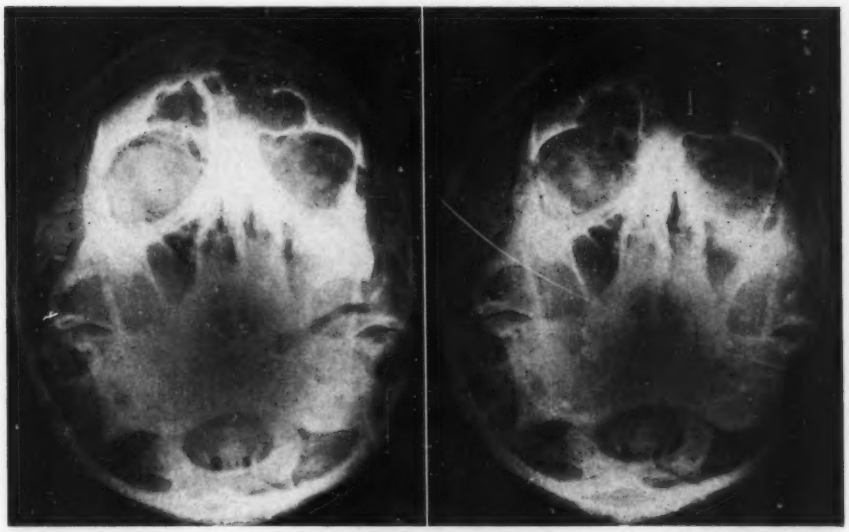


Fig. 2 (Case 1)—Left: Preoperative Waters' view of facial bones reveals medial dislocation of zygoma at zygomaticofrontal suture line and considerable medial rotation of arch, with rather apparent dehiscence at this point. Note fracture through infraorbital ridge and fracture of lateral wall of sinus medially displaced, continuous with zygomatic process of maxilla. Left antrum was cloudy because of hemorrhage. Right: Postoperative Waters' view reveals realignment of zygomaticofrontal suture line, infraorbital ridge and lateral wall of sinus and re-established arch continuity. Note restored symmetry of bony outline when compared with normal side

CASE 2—A soldier was admitted to the U. S. Army Hospital, Landstuhl, Germany, on February 14, 1955, after incurring injuries in an automobile accident the day before admission. There was a pronounced depression of the right zygoma, periorbital edema, no diplopia and a hypesthesia of the infraorbital region. A palpable depression of the arch and some crepitation were noted. There was some pain and difficulty in opening the mouth. Roentgenograms revealed a depressed fracture of the zygomatic arch (Fig. 3, above left) and separation of the zygomaticofrontal suture line with medial displacement, a fracture through the infraorbital ridge and the anterolateral wall of the antrum (Fig. 3, above right).

Elevation and reduction was achieved under general anesthesia on February 16. The modified sound was used for the operation (Fig. 3, below).

CASE 3—A Canadian airman was admitted to the U. S. Army Hospital, Landstuhl, Germany, on June 1, 1955, with a depressed fracture of the right zygoma. The injury was incurred on May 26, 1955, when the patient collided with another player in a ball game. Roentgenograms revealed a depressed fracture of the zygomatic arch with overlapping and a fracture at the zygomaticofrontal suture line with slight lateral displacement of the

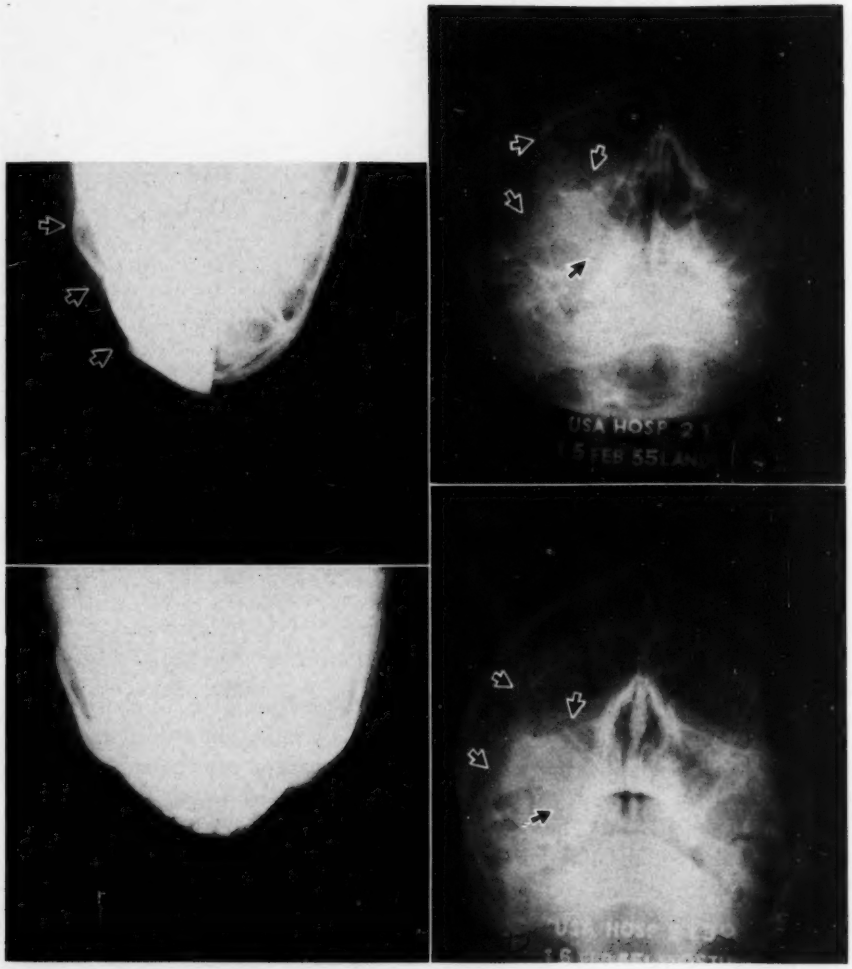


Fig. 3 (Case 2)—Above left: Preoperative view of zygomatic arch. Note fracture depression of arch. Above right: Preoperative Waters' view. Note apparent fracture dislocation of zygoma at zygomaticofrontal suture line with medial displacement, fracture through infraorbital ridge and anterolateral wall of antrum. Below left: Postoperative arch view. Note normal alignment of fragments of arch. Below right: Postoperative Waters' view. Note resultant normal alignment at zygomaticofrontal suture line, infraorbital ridge and arch

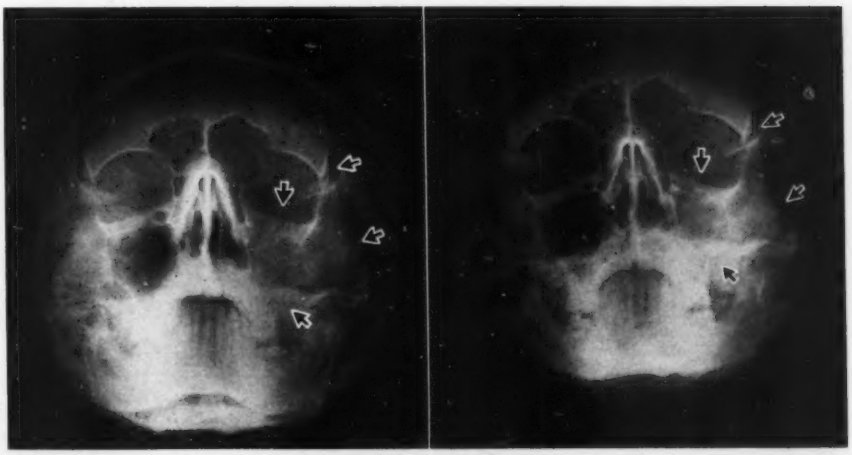


Fig. 4 (Case 3)—Left: Waters' view reveals fracture at zygomaticofrontal suture line with slight lateral displacement of frontosphenoidal process, the infraorbital ridge, arch and lateral wall of antrum. Right: Waters' view reveals postreduction symmetry

frontosphenoidal process, the infraorbital ridge and lateral wall of the antrum (Fig. 4, left). The operation was performed on June 2, under general anesthesia. The modified sound was used. Postoperative roentgenograms confirmed the operative impression of restoration of normal symmetry (Fig. 4, right).

CASE 4—A soldier was admitted to the U. S. Army Hospital, Landstuhl, Germany, on May 29, 1955, a few hours after incurring a fracture of the left zygoma. The patient stated that he had been struck by an unknown assailant while sitting in a *Gasthaus*. Roentgenograms revealed a pronounced depression of the zygomatic arch (Fig. 5, above left) in addition to medial displacement of the zygoma, depression of the infraorbital ridge and lateral wall of the antrum (Fig. 5, above right). The reduction was accomplished under general anesthesia on June 1, using the modified sound. Postoperative roentgenograms showed good reduction of all fractures (Fig. 5, below).

CASE 5—The wife of a Canadian airman was admitted to the U. S. Army Hospital on June 27, 1955. In an automobile accident, she had incurred a depressed fracture of the right zygoma, compounded by a laceration of the lateral aspect of the eyebrow. There was a pronounced depression, diplopia and crepitation. Preoperative roentgenographic examination revealed medial displacement of the zygoma with rotation and dehiscence of the infraorbital ridge (Fig. 6, left). The reduction was accomplished under general anesthesia on June 28 with the use of the modified sound. Postoperative films revealed adequate reduction of all fractures (Fig. 6, right).

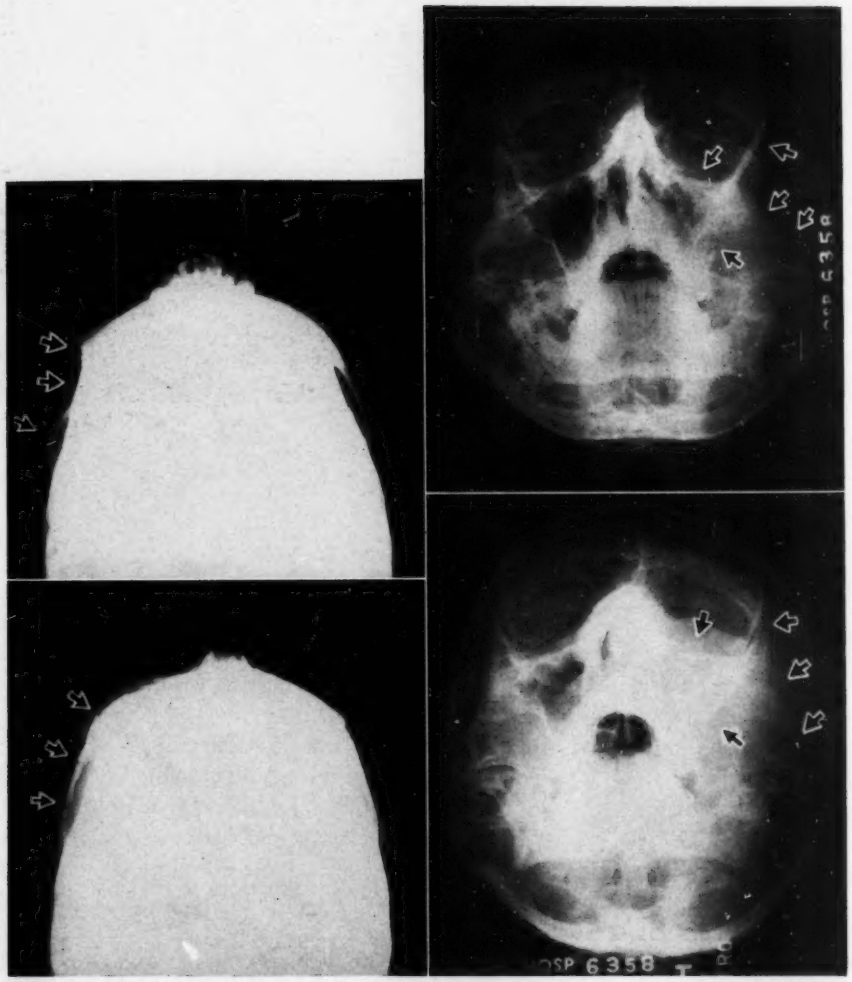


Fig. 5 (Case 4)—Above left: Preoperative arch view reveals pronounced depression of arch. Above right: Preoperative Waters' view shows medial displacement of zygoma at zygomaticofrontal suture line, depression of infraorbital ridge, fracture of arch and pronounced depression of lateral wall of antrum. Below left: Note normal alignment after reduction. Below right: Postoperative Waters' view reveals relatively normal alignment

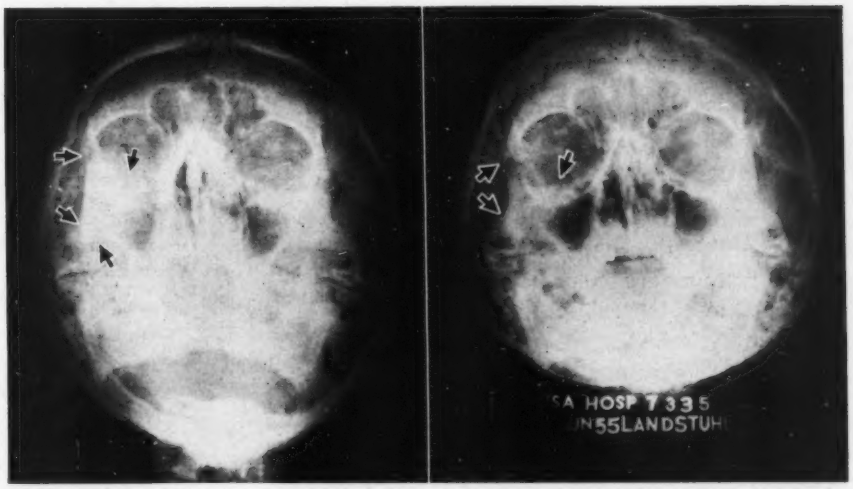


Fig. 6 (Case 5)—Left: Waters' view reveals medial displacement of zygoma at zygomaticofrontal suture line, dehiscence of infraorbital ridge and lateral rotation of anterior aspect of zygoma. Right: Postoperative Waters' view. Note relatively normal alignment after reduction

■ *Summary*

The metal urethral sound, which has been used as an instrument for reduction of zygomatic fractures for years, was felt to have certain inherent deficiencies when used for this purpose. Two welded metal attachments were added to overcome these deficiencies. The modified sound has been used on a series of 88 patients and is considered to have distinct advantages as an addition to the existing armamentarium. It is used, by the author, only with the intraoral buccal approach, and only with pull force. There is no need for levering action with possible damage to the fulcrum area. Five cases of various types of zygomatic fractures in which this instrument was used are reported.

■ The Research Worker

The research worker deals with the undefined boundaries of knowledge and with the frayed edges of sound information. He does not march with the procession, but he must do lonely outpost and scouting duties.—*Theobald Smith.*

Oral leukoplakia: review of the literature

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A partial review of the literature has been made to determine the status of leukoplakia as a precancerous lesion, and to establish the areas of this subject that are in need of further study.

The material pertaining to the relation of oral leukoplakia to cancer is difficult to review for the following reasons: multiple definition and the use of synonyms for the disease;[1] the wide range of opinions as to etiology, treatment and prognosis of the lesion when discussed by the various authors; the large number of articles written which are based on opinion but subsequently quoted as fact; information on the subject often obscured by misleading or mistitled articles; the use of terminology which is not correct histologically; a dearth of factual material in the dental literature and the fact that the disease in other areas of the body seemingly has been considered as a separate entity.

■ *Incidence of Carcinoma in Leukoplakia*

In 1948, Bernier and Ash[2] described leukoplakia as:

> . . . being characterized by variable degrees of keratosis, either as hyperkeratosis, acanthosis, and inflammatory exudate in the corium. The important feature, however, is the malignant type of dyskeratosis, which may be so pronounced as to make differentiation from carcinoma difficult.

Kronfeld[3] states:

> . . . leukoplakia is a thickening of the epithelial layer with parakeratosis or hyperkeratosis; and that from 20% to 30% of all oral squamous cell carcinomas develop from pre-existing leukoplakia patches.

The total number of cases examined is not recorded.

Cahn[4] stated as follows:

> On a histological basis leukoplakia can be divided into two entities, the distinguishing factor between the two being the formation of a well marked horny layer. These entities are keratosis and epithelial hyperplasia. Both histological types of leukoplakia may be found in the same specimen. A transition from keratosis to marked epithelial hyperplasia will be seen, with the loss of the hornified zone and a great increase in the size of the prickle cell zone. This type of transformation is a malignant trend on the part of the tissues.

Bernier[5] stated:

> An accurate definition of leukoplakia is difficult unless definite diagnostic criteria are established. More important, perhaps, are the degree and distribution of the changes in the tissue. Some of these changes are hyperkeratosis, parakeratosis, dyskeratosis, acanthosis, hydropic degeneration and accompanying inflammatory

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1. Schaffer, J. Clinical pathology of the tongue. Oral Surg., Oral Med. & Oral Path. 5:87 Jan. 1952.

2. Bernier, J. L., and Ash, J. E. Leukoplakia. In Atlas of dental and oral pathology, ed. 4. Washington, D. C., Registry Press, 1948.

3. Boyle, P. E., editor. Leukoplakia. In Kronfeld's Histopathology of the teeth, ed. 4. Philadelphia, Lea & Febiger, 1955.

4. Cahn, L. R. Leukoplakia. Pathology of oral cavity. Baltimore, Williams & Wilkens Co., 1941, p. 197.

5. Bernier, J. L. Manual for the differential diagnosis of oral lesions. St. Louis, C. V. Mosby Co., 1942, p. 38.

changes in the corium. In advanced lesions the possibility of early malignancy is considerable.

Bernier[6] in 1949 made a more positive statement:

Early dyskeratosis must be present if the lesion is to be judged as potentially malignant. Epithelium that is undergoing simple hyperplasia and shows inflammatory reaction as a result of external trauma may or may not be transformed into a frankly malignant lesion. Unless the trend in this direction can be demonstrated histologically, there is no justification for considering a case of leukoplakia as malignant. The feature that indicates potential malignancy, although difficult to evaluate, can probably be best described as early disruption in the maturation of the epithelium or dyskeratosis.

In an analysis of over 400 cases of intraoral squamous cell carcinoma in 1954, Tiecke and Bernier[7] included the following statement in their article:

Some authors regard leukoplakia as the chief precursor of carcinoma in the oral cavity. However, it is obvious that many investigators make the diagnosis of leukoplakia on clinical appearance (a white patch) rather than on the histological features of the lesion. In this report the term pachyderma oralis is used to differentiate the lesion from leukoplakia because dyskeratosis, which is considered a prerequisite for the pathological diagnosis of leukoplakia, could not be demonstrated in any of the cases in which a white patch represented the initial lesion.

Figure 1 illustrates pachyderma oris. Differentiation beween pachyderma oris and leukoplakia is demonstrated in Figures 2 and 3.

Kollar and others[8] suggested that,

6. Bernier, J. L. Histologic and clinical features of leukoplakia. Bul. U.S. Army Med. Dept. 9:621 July 1949.

7. Tiecke, R. W., and Bernier, J. L. Statistical and morphological analysis of four hundred and one cases of intraoral squamous cell carcinoma. J.A.D.A. 49:684 Dec. 1954.

8. Kollar, J. A., and others. Leukoplakia. J.A.D.A. 49:538 Nov. 1954.

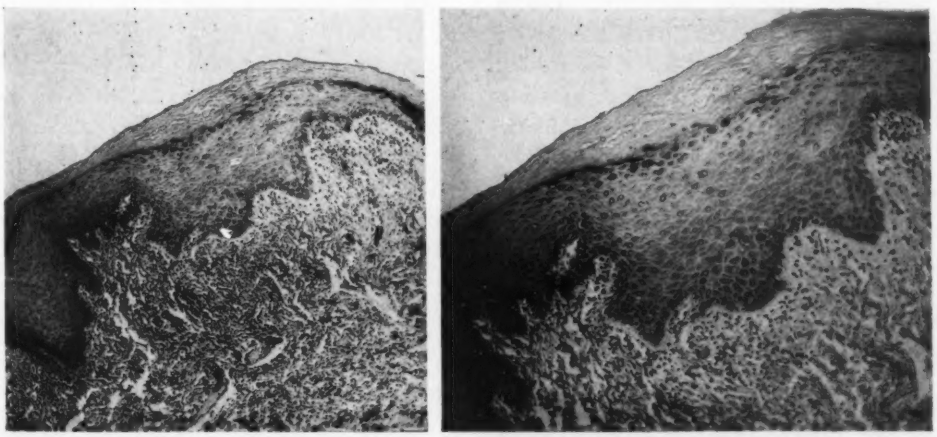

Fig. 1—Pachyderma oris in oral mucosa. Section is from white patch on alveolar ridge of middle-aged man. Surface shows increased thickness of keratin (hyperkeratosis). Prickle cell layer is of approximately normal thickness with orderly arrangement of cells. Basal layer is well-demarcated. Right: Higher magnification of same specimen. Only significant pathologic change is increased thickness of keratinized layer. (Illustration courtesy of Armed Forces Institute of Pathology)

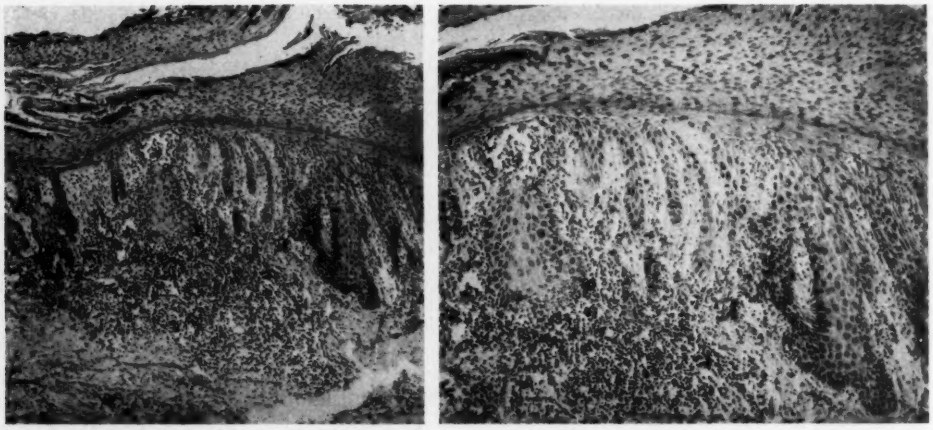

Fig. 2—Left: Leukoplakia in lower lip. Specimen from lip-shave operation on middle-aged man. Patient had diffuse whitish lesion of lower lip with cracking and scaling. Microscopic examination shows severe parakeratosis of surface. Note irregular acanthosis and dyskeratosis. Right: Higher magnification of same specimen. Dyskeratosis evident by loss of normal arrangement of epithelial cells, hyperchromatism, variation in cell size and increased mitosis. Invasion, however, not demonstrated in specimen. Round-cell infiltrate is present in lamina propria. (Illustration courtesy of Armed Forces Institute of Pathology)

"The use of the term 'leukoplakia' should be discontinued as a diagnosis." They also gave a simple classification for maturation disorders of the oral mucous membrane. As indicated in their article the advantages of their classification are as follows: (1) a discussion of what leukoplakia is microscopically is eliminated; (2) dissension as to whether it is benign, cancerous or precancerous can be avoided and (3) most important of all, a basis for a rational plan of case management is provided. The simple classification for maturation disorders of the oral mucous membranes based on a biopsy specimen is as follows:

1. Hyperplasia.
2. Hyperkeratosis.
3. Hyperkeratosis and inflammation.
4. Hyperkeratosis, dyskeratosis and inflammation.
5. Intraepithelial cancer.
6. Epidermoid carcinoma.

Sturgis and Lund[9] in a study of 298 patients with leukoplakia found that 12 per cent developed malignancy during a five year period of observation.

Lain[10] indicated in a review of 82 cases of oral leukoplakia that cancer subsequently developed on the lower lip of two of the patients. Both of Lain's patients were full-blooded Indians. He did not indicate that a biopsy was used for any of his diagnostic findings, nor did he mention the duration of his observations.

Boyle[11] found, "In 27 instances of leukoplakia diagnosed clinically, and then examined microscopically, one lesion had

9. Sturgis, S. H., and Lund, C. C. Leukoplakia buccalis and keratosis labialis. New England J. Med. 210:996, 1934.

10. Lain, E. S. Lesions of oral cavity caused by physical and by physicochemical factors. Arch. Dermat. & Syph. 41:295 Feb. 1940.

11. Boyle, P. E. Differential diagnosis of soft tissue lesions of the mouth with a discussion of biopsy procedures. Oral Surg., Oral Med. & Oral Path. 7:507 May 1954.

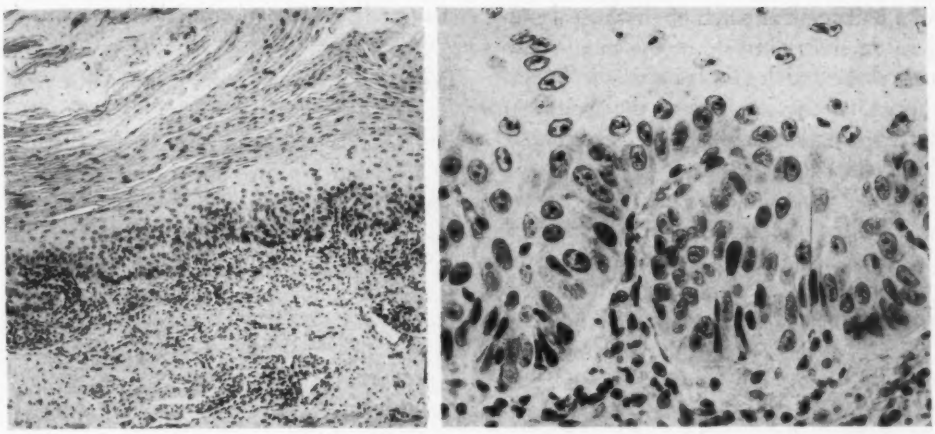

Fig. 3—Left: Leukoplakia of lower lip. Specimen from localized white patch on lower lip. Surface covered by greatly thickened layer of parakeratin. Acanthosis not a feature; however, dyskeratosis is evident. Right: High magnification of same specimen showing details of dyskeratotic epithelium. Note variations in cell size, hyperchromatism, abnormal cell structure and loss of orderly arrangement. Basal layer is intact, however; no evidence of invasion. (Illustration courtesy of Armed Forces Institute of Pathology)

already undergone malignant, neoplastic change."

Burford[12,13] reported a case of carcinoma of the hard palate and another of the lip after the pathology report had indicated leukoplakia of those areas.

In an analysis of 636 patients treated for cancer of the lip, Schreiner and Christy[14] found a history of cracked or chapped lips or chronic fissure in 389 of the patients. One hundred and ninety-seven of the patients had definite histories or signs of what was interpreted as keratosis. There were only 15 instances of leukoplakia. In this article no explanation was made as to what histological differences were present that differentiated the keratosis of the lip from leukoplakia.

In an analysis of statistics compiled from hospital records that extended from 1932 to 1946, Hobaek[15] reviewed 1,272 instances of oral cancer. He found that 176 instances or 16 per cent developed on a basis of pre-existing leukoplakia. In that same study he found 77 cases of uncomplicated leukoplakia, by which it is assumed is meant that the condition cleared completely after one episode of treatment.

Hazen and Eichenlaub[16] observed two instances of cancer that developed in an area of leukoplakia in ten patients that were under treatment at the time.

Fleming[17] reported early carcinoma

12. Burford, W. N. Symposium of eighteen cases of benign and malignant lesions of the oral cavity, from the Ellis Fischel State Cancer Hospital, Columbia, Missouri. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 33:1 Jan. 1947.

13. Burford, W. N. Dentist's responsibility in oral tumors. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 29:612 Nov. 1943; correction 30:56 Jan. 1944.

14. Schreiner, B. F., and Christy, C. J. Results of irradiation treatment of cancer of lip; analysis of 636 cases from 1926-1936. Radiology 39:293 Sept. 1942.

15. Hobaek, A. Leukoplakia oris. Acta odont. Scandinav. 7:61 May 1946.

16. Hazen, H. H., and Eichenlaub, F. J. Leukoplakia buccalis. J.A.M.A. 79:1487 Oct. 28, 1922.

17. Fleming, W. E. Early carcinoma in a leukoplakial patch. Austral. J. Den. 51:359 Nov. 1947.

in a leukoplakial patch of the buccal mucosa on the right side of the mouth and uncomplicated leukoplakia of the buccal mucosa of the left side of the mouth in the same patient. Biopsy was used in making both diagnoses.

Carr[18] reported a case where there was a lapse of four years from the initial diagnosis of leukoplakia until a diagnosis of squamous cell carcinoma of the same area was made. At the time of the initial diagnosis of leukoplakia, the patient stated the lesion was of nine years' duration. If this was true, it would mean that there was a 13 years' time lapse from the beginning of tissue changes until carcinoma was diagnosed.

In 1928 Prinz[19] stated that "the time which elapses before cancer develops upon a leukoplakic base will range from 2 to 20 years."

In a statistical analysis of 100 cases of oral leukoplakia, McKown[20] found that malignant degeneration occurred in 30 per cent of the cases. The average duration of the leukoplakia was 1.4 years.

Sarnat and Schour[21] mention a case reported in which an initial diagnosis of leukoplakia of the tongue was made from biopsy, and 18 months later biopsy showed carcinoma.

The need and importance of biopsy was pointed out in a rather unusual case by Thoma.[22] A clinical diagnosis of carcinoma was made; leukoplakia was proved by biopsy.

In a study of 2,300 instances of all types of oral lesions, McCarthy[23] found that leukoplakia was present in 14 per cent. He also reported:

> The role of leukoplakia as a forerunner of cancer cannot be determined by this statistical study, but it can be stated that carcinoma frequently arises from leukoplakia lesions found in syphilitic atrophic glossitis.

McCarthy, however, did not give the percentage of instances of carcinoma after leukoplakia had been observed in biopsy.

Ziskin[24,25] gave impetus to dental research by his work on the effects of hormones on gingival and oral mucous membranes. He found that hormonal secretions are a potent factor in the etiology of certain gingival diseases. When estrogen was injected into monkeys or women, hyperkeratinization resulted in areas of the oral mucosa that normally have keratin. Keratin was also seen on the surface of the oral mucous membranes where none is found normally. It would be of interest to know if leukoplakia was ever produced in the estrogenic series and if so, whether the experiment could be continued to the point where carcinoma is produced in the leukoplakia areas.

Wolbach[26] in 1937 found that the pathology of vitamin A deficiency indicates that the seat of the physiologic disturbance is in the epithelial cell and that even though the chemical role of the cell is suppressed, the proliferative powers are not lost. In vitamin A deficiency the se-

18. Carr, M. W. Squamous cell carcinoma of the tongue as a sequel to leukoplakia. J. Oral Surg. 6:78 Jan. 1948.

19. Prinz, H. Leukoplakia oris—a clinical study. D. Cosmos 70:663 July 1928.

20. McKown, J. L., Jr. Leukoplakia, a precancerous condition; statistical analysis of 100 cases. (Abst.) Bul. U.S. Army Med. Dept. 9:620 July 1949.

21. Sarnat, B., and Schour, I. Oral and facial cancer. Chicago, Yearbook Publishers, 1950.

22. Thoma, K. H. Papillomatosis of the palate. Oral Surg., Oral Med. & Oral Path. 5:214 Feb. 1952.

23. McCarthy, F. P. Clinical and pathologic study of oral disease based on 2,300 consecutive cases. J.A.M.A. 116:16 Jan. 4, 1941.

24. Ziskin, D. E. Effects of certain hormones on gingival and oral mucous membranes. J.A.D.A. & D. Cosmos 25:422 March 1938.

25. Ziskin, D. E. Effects of male sex hormone on gingivae and oral mucous membranes. J. D. Res. 20:419 Oct. 1941.

26. Wolbach, S. B. Pathologic changes resulting from vitamin deficiency. J.A.M.A. 108:7 Jan. 2, 1937.

quences are atrophy of the epithelium concerned, and the substitution for it by a stratified keratinizing epithelium. An invariable sequence in pathology is that a break in the continuity of a tissue is followed by a reparative proliferation. In vitamin A deficiency the basal cells normally concerned in maintaining the integrity of epithelium respond by active mitotic division. As the basal cells have distribution in all nonstratified epithelium, the next effect of the deficiency after atrophy is the appearance of scattered areas of proliferative activity beneath the original epithelium. The new (reparative) cells by their continued growth undermine and replace the original epithelium and, regardless of previous function and structure of the region, develop into a stratified keratinizing epithelium.

Kreshover's[27] experiments with mice substantiate the contention of some authors that vitamin B deficiency increases the susceptibility of tissue to smoke irritant whereas gonadectomy establishes an appreciable degree of resistance.

In describing some of the pathology that resulted in his work with the hamster, Salley[28] used the term *"in situ."* This term is used freely in the medical literature in regard to uterine cancer but was not found in the dental literature reviewed. According to Carter,[29] "carcinoma *in situ*" is a term which should be applied to a microscopic picture of the surface cervical epithelium in which the individual cells through the full thickness of the epithelial layer have the same characteristics as those of invasive cancer. This is stage O in the International Classification of Cervical Cancer. Apparently *"in situ"* is intended to mean that the basal membrane is intact and the underlying connective tissue has not been invaded. Kollar[8] has recommended calling this stage of epithelial maturation disorder of the oral mucous membrane "intraepithelial cancer."

In none of the articles reviewed was leukoplakia reported as being produced experimentally deliberately. In Salley's experiment[28] with the Syrian hamster he described four degrees of change histologically: hyperplasia, benign papilloma, squamous cell carcinoma *in situ* (preinvasive) and squamous cell carcinoma with local invasion and metastasis. In the description of the benign papilloma stage of the production of cancer the following appeared:

> Fig. No. 3 is a section of pouch wall with epithelial hyperplasia, hyperkeratosis, parakeratosis, acanthosis, and some downward proliferation of the epithelium into the submucosa. However, the epithelium is still well stratified, and although there appears to be independent nests of epithelial cells at the bottom center of the photograph, adjacent serial sections revealed these to be connected to the large mass of cells. There was no evidence of cell or nuclear atypicality.

It would be of interest to know at this point whether if all irritants had been removed the lesion described above would have progressed to cancer *in situ*, and eventually to invasive carcinoma with metastasis.

In the treatment of oral leukoplakia, cessation of smoking has been accepted as a method of reversing the process. The literature contains abundant evidence to

27. Kreshover, S. J. Effect of tobacco on epithelial tissues of mice. J.A.D.A. 45:528 Nov. 1952.

28. Salley, J. J. Experimental carcinogenesis in the cheek pouch of the Syrian hamster. (Abst.) J. D. Res. 32:679 Oct. 1953.

29. Carter, B. N., editor. Monographs on surgery. New York, Thos. Nelson & Sons, 1950, p. 3.

substantiate this method of treating clinical leukoplakia.

In reviewing the various hypotheses on how the carcinogenic process takes place, Greenstein and Haddow[30] discuss the following theories: field effect, single cell origin, virus, heredity, initiating and promoting action as independent stages of carcinogenesis, and others. They state:

> At the present time the virus hypothesis has become a crucial point in discussions on the etiology of cancer. Whether one likes it or not, it offers the best explanation for malignancy; yet now as always, it has the united forces of the greater part of oncologists ranged against it. Much research remains to be done before we know the ways in which nucleoproteins can become parasitic, which is probably the basis of carcinogenesis. But at the moment this is speculative, and it may be asked whether it may not be precisely a foreign origin which endows the nucleoproteins with their independence, or in other words the autonomy which makes them the agents of cancer.

If leukoplakia is considered as a pathological process, the various hypotheses for cancer might well be applied to leukoplakia.

■ *Diagnosis*

An effort was made by Montgomery and von Haam[31] to discover typical leukoplakia cells from cytologic smears. The Papanicolaou method was used in this study of ten patients with oral leukoplakia. Although the smears showed some evidence of increased keratinization taking place, no cytologic characteristics were found suggesting premalignant change. "No typical leukoplakia cell could be discovered and even detailed cytologic study failed to reveal any abnormality in the nucleus or nucleolus suggestive of any change in the cell structure."

Piro and others[32] made Gelfoam sponge biopsies from 11 patients proved to have oral carcinoma but found that the method was unsatisfactory for making a diagnosis of cancer. Pomeranz and Stahl[33] used the smear technic in a similar test of eight oral malignancies diagnosed as such from previous biopsy. Of the eight tested, six were positive, one negative and one suspicious.

In a study undertaken by Stone[34] using the direct smear technic, investigation was made of the keratinization of the oral epithelium. For this study 50 men were used whose ages ranged from 60 to 100 years. Stone concluded that there is a definite decrease of gingival keratinization in aged men, and that in the aging process, the tissue of the oral cavity lacks a protective layer of keratinized cells. This lack renders the tissue more susceptible to mechanical, chemical or bacterial irritation.

Martin and co-workers[35] in another investigation of 100 men and 100 women all over 40 years of age examined these patients for oral leukoplakia. None of this group had any intraoral complaint. In the men 50 per cent had some degree of leukoplakia whereas the group of women showed some evidence of oral leukoplakia. The presence of some degree of leuko-

30. Greenstein, J. P., and Haddow, A., editors. Advances in cancer research, vol. 2. New York, Academic Press, Inc., 1954, p. 159.

31. Montgomery, P. W., and von Haam, E. Study of the exfoliative cytology of oral leukoplakia. J. D. Res. 30:260 April 1951.

32. Piro, J. D., and others. Gelfoam sponge biopsy in the diagnosis of oral carcinoma. Oral Surg., Oral Med. & Oral Path. 7:417 April 1954.

33. Pomeranz, M. J., and Stahl, S. S. Correlative study of cyto-diagnosis and biopsy. Oral Surg., Oral Med. & Oral Path. 6:1026 Aug. 1953.

34. Stone, A. Keratinization of the human oral mucosa in the aged male. J. D. Med. 8:69 April 1953.

35. Martin, H.; MacComb, W. S., and Blady, J. V. Cancer of lip. Ann. Surg. 114:226 Aug.; 341 Sept. 1941.

plakia in the oral membranes after middle age cannot be considered abnormal. In the majority of cases it simply represents a response of the membranes to mild chronic irritation and as such, is a part of the natural aging process comparable to the almost universal mild senile changes including keratosis of the skin which begin at or soon after middle age.

Papanicolaou[36] listed the rapid advances made in exfoliative cytology. Unfortunately no mention was made of the use of this test on cells of the oral cavity. The Papanicolaou technic of exfoliative cytology diagnosis has not been as reliable as the profession had hoped for.[31-34]

Ronchese[37] found that the Wood light was of little value in early diagnosis of carcinoma and that in leukoplakia, the more severe and keratotic is the lesion, the whiter the lesion will appear under the Wood or black light.

■ *Treatment*

In a study of the relation between dietary deficiency and the occurrence of papillary atrophy of the tongue and oral leukoplakia, Abels and co-workers[38] found that daily doses of brewer's yeast resulted in remission of the leukoplakia in from two to ten months.

Martin and Koop[39] wrote that they have observed instances of striated leukoplakia of the oral mucous membrane which responded to vitamin B therapy.

Schour and Massler[40] call attention to the possibility that vitamin A deficiency may also play a role in oral cancer in view of the characteristic metaplasia of epithelial cells. Vitamin A deficiency may result in hyperkeratotic changes in the oral mucosa. Usually, however, the clinical symptoms of the vitamin deficiencies disappear on the addition of the needed vitamin to the diet.

Bacchi[41] reported a cure of leukoplakia with massive doses of both vitamin A and B.

In a review of 330 patients' records at the Veterans Administration Hospital, Hines, Ill., Gibbel and co-workers[42] reported the complete disappearance of leukoplakia in ten patients treated with brewer's yeast.

Burket[43] stated, however, "that the general lack of response to therapeutic doses of vitamin A of patients with oral leukoplakia suggests that vitamin A is not an important or common systemic predisposing factor." Madden[44] indicated that he had found that vitamins A and B were of little value in the treatment of leukoplakia.

In 1933 Hollander and others[45] recommended elimination of all possible irritations in leukoplakia of the oral mucosa, and if it did not clear up in 14 days, removal by cautery. Howell[46] recommended the shaving operation in recur-

36. Papanicolaou, G. N. Recent trends in cytologic diagnosis. New York J. Med. 53:39 Jan. 1, 1953.

37. Ronchese, F. Fluorescence of cancer under the Wood light. Oral Surg., Oral Med. & Oral Path. 7:967 Sept. 1954.

38. Abels, J. C., and others. Relationship between dietary deficiency and occurrence of papillary atrophy of tongue and oral leukoplakia. Cancer Res. 2:381 June 1942.

39. Martin, H., and Koop, C. E. Precancerous mouth lesions of avitaminosis B; their etiology, response to therapy and relationship to intra-oral cancer. Am. J. Surg. 57:195 Aug. 1942.

40. Schour, I., and Massler, M. Effects of dietary deficiencies upon the oral structures. J.A.D.A. 32:714 June 1945.

41. Bacchi, J. G. Leukoplakia treated with vitamins A and B. D. Survey 22:1306 July 1946.

42. Gibbel, M. I.; Cross, J. H., and Ariel, I. M. Cancer of the tongue; review of 330 cases. Cancer 2:411 May 1949.

43. Burket, L. W. Oral leukoplakia. In oral medicine; diagnosis and treatment, ed. 2. Philadelphia, J. B. Lippincott Co., 1952, p. 133.

44. Madden, J. F. Leukoplakia buccalis. In Current therapy. Philadelphia, W. B. Saunders Co., 1952, p. 558.

45. Hollander, L.; Permar, H. H., and Shonfield, Leo. Leukoplakia of the oral mucosa. J.A.D.A. 20:41 Jan. 1933.

46. Howell, J. B. Cancer and precancer of the skin. In Current therapy. Philadelphia, W. B. Saunders Co., p. 514.

ring leukoplakia of the lower lip. This operation consists of the excision of the vermilion border of the lower lip, which eliminates the potential cancer-bearing area. Herold[47] suggested either the scalpel excision or cautery if the lesion does not regress after elimination of all etiologic factors.

Martin[48] pointed out the important role that the dentist can play in early diagnosis of mouth cancer:

> Since leukoplakia occurs as a response of mucous membranes to irritation, and the etiology is identical with the main factors of oral cancer, its presence should serve as a warning to the patient that he is more likely to develop a malignant tumor of the mouth than the average person.

In *Monographs on Surgery*[29] leukoplakia of the mucous membrane of the uterus was described as follows: "Leukoplakia, this is a term referring to hyperkeratinized, thickly cornified plaque which overlies immature and active epithelium usually associated with some abnormal basal cell hyperactivity." Although dyskeratosis is not included in this description of uterine leukoplakia, it is assumed that the phrase "abnormal basal cell hyperactivity" implies dyskeratosis. If this is true, the question arises whether the actual histologic specimen differs so greatly from the oral leukoplakia specimen as to make it a separate diagnostic entity.

■ *Incidence of Leukoplakia in Carcinoma*

In 103 cases of cancer of the floor of the mouth, Martin and Sugarbaker[49] reported "leukoplakia present in 25% of the cases. This incidence is less than in cancer of the tongue, 46%, or of the cheek, 70%. According to their statistics, these findings indicate that chronic irritation is probably a less prominent etiological factor in the floor of the mouth than in other forms of oral cancer."

In a series of 375 cases of lip cancer, Martin and others[35] found leukoplakia present on the lips, tongue and cheek in about 28 per cent of the patients.

Archer and Morris[50] in a report of a survey of 203 patients with oral carcinoma stated that leukoplakia was found in 32 patients or 15.8 per cent of the total group. They also recorded that of the 203 patients, 57 per cent gave no evidence of any one of the possible contributing factors such as syphilis, leukoplakia, heavy smoking, trauma, or irritation from various sources.

Sharp and Spickerman[51] found leukoplakia present in 40 per cent of one group of 27 patients with lingual cancer, and in a group of 54 veterans with lingual cancer, 37 per cent had leukoplakia.

Bernier and Clark[52] reviewed 317 cases of carcinoma of the lip and found that the initial clinical feature ranged from an ulcer to keratosis. The terminology used in the clinical abstracts made it difficult in determining the exact meaning of the descriptions of the various lesions. About 40 of the patients in this series had a secondary diagnosis of leukoplakia. The

47. Herold, W. C. Leukoplakia buccalis. In Current therapy. Philadelphia, W. B. Saunders Co., 1952, p. 557.

48. Martin, H. E. Mouth cancer and the dentist. J.A.D.A. 33:845 July 1946.

49. Martin, H. E., and Sugarbaker, E. D. Cancer of the floor of the mouth. Surg., Gyn. & Obst. 71:347 Sept. 1940.

50. Archer, W. H., and Morris, R. D. Survey of oral carcinoma at the Columbia-Presbyterian Medical Center. Am. J. Orthodont. & Oral Surg. (Oral Surg. Sect.) 32:338 June 1946.

51. Sharp, G. S., and Spickerman, H. D. Cancer of tongue. Am. J. Roentg. 57:181 Feb. 1947.

52. Bernier, J. L., and Clark, M. Carcinoma of lip; preliminary analysis of 330 cases with follow up of from 3 to 36 years. Mil. Surgeon 102:474 June 1948.

word keratosis was frequently used to designate lesions which in all probability were leukoplakia.

The incidence of leukoplakia reported as being present at the time of diagnosis of oral cancer ranges from 15.8 per cent[50] to 70 per cent.[49] In comparing these figures with the incidence of leukoplakia as a precancerous lesion, the percentage ranged from 12 per cent[9] to 30 per cent.[20]

■ *Discussion*

Most of the writers who express an opinion on the treatment of leukoplakia agree that silver nitrate, radium or roentgen ray are not very satisfactory methods of treatment. They also are fairly much in agreement on the cause of leukoplakia's being some irritation, usually in the form of tobacco or dental abnormalities. The galvanic theory has disappeared from the literature on the cause of leukoplakia.

Actually little factual specific information is available to the dental profession on the subject of leukoplakia. The trial and error method is still apparently the principal approach to the problem. A patient with leukoplakia of the oral cavity can stop smoking and may be rewarded with complete disappearance of the lesion. The irritant to be found in the edentulous patient who doesn't smoke or use alcohol, is nonsyphilitic, doesn't have dentures and yet has oral leukoplakia is not known.

■ *Summary and Conclusions*

1. The majority of writers indicate that they believe leukoplakia is a precancerous lesion.

2. There is not a large volume of conclusive evidence that leukoplakia is a precancerous lesion.

3. Many leukoplakia lesions are being diagnosed and treated from clinical observations without benefit of biopsy.

4. There is a need for standardization of terminology and method of diagnosis.

5. If dentists are using a follow-up system of their patients with leukoplakia they are not reporting the results in the dental literature.

6. The areas of the subject in need of further research are as follows:

a. Review of the literature as to the comparative value of the present methods of treatment.

b. Case history study of hospital records of oral cancer in those patients with an initial diagnosis of leukoplakia.

c. Experimental study to determine whether leukoplakia will regress spontaneously when the causative agent is removed.

d. Experimental study to determine whether leukoplakia can be transplanted to another animal.

e. Experimental study to determine what abnormal change takes place in the individual cells in an area of leukoplakia.

7. The acceptance by the dental profession of Kollar's[8] classification for maturation disorders in the epithelium of the oral mucous membrane would afford better communication of observations by clinicians and authors discussing this pathologic condition.

8. The acceptance of dyskeratosis,[5] as a prerequisite for the pathological diagnosis of leukoplakia might well afford a basis for mutual understanding of an oral condition about which so little is actually known but about which so much has been written.

Carcinoma, sarcoma and melanoma: their diagnosis and treatment

■ *Kurt H. Thoma, D.M.D., F.D.S., R.C.S., Eng., Brookline, Mass.*

Today the dentist is well aware of his responsibility in the diagnosis of cancer. Most dentists recognize suspicious oral lesions, and some even take biopsies to ascertain their nature. The discovery of a malignant lesion, however, brings with it the obligation of advising the patient regarding treatment. Here the dentist often fails. He may be in doubt as to the nature and extent of the malignancy. Often valuable time is lost because he hesitates to refer his patient when he is uncertain as to which type of treatment promises the best result.

The most important decision to be made is whether the patient should be given surgical treatment or irradiation. A combination of the two is rarely advocated today although in selected cases it may be advantageous. Usually the disease is either amenable to surgery or it may be radiocurable, or at least controlled for a time by palliative roentgen ray treatment. What are the factors which help to make the decision?

■ *Diagnosis and Treatment Plan*

To make a comprehensive study of a patient with malignant disease and to decide on the best type of treatment in each individual instance requires a great deal of experience. Few dental or medical practitioners are sufficiently experienced in the treatment of malignant disease to advise the occasional patient they may see. This is easily understandable because in cancer clinics such as are established in most of the larger civilian hospitals as well as the veterans' hospitals, a group of representatives from various departments discuss the diagnosis, pathology, treatment and postoperative care of each individual patient. This group includes men from general surgery, the various specialties including oral surgery, pathology, radiology, prosthetic dentistry and other departments dealing with rehabilitation of the patients who have had important parts of their bodies removed, to effect a cure of the fatal disease.

The following factors should be evaluated carefully and related to the treatment:

TYPE OF MALIGNANT GROWTH—By far the largest percentage of oral malignant growths are the various types of carcinoma. Differentiation must be made, however, between squamous cell, transitional cell and adenocarcinoma. In addition, great variations in rapidity of growth, cell differentiation and invasiveness of the tumor cells must be recognized. A carcinoma may be an ulcerating, deeply infil-

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Fig. 1—Squamous cell carcinoma, ulcerating type, grade II

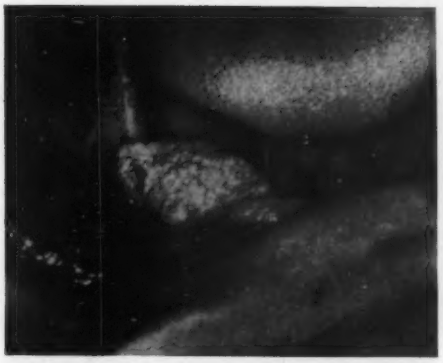

Fig. 2—Squamous cell carcinoma, verrucous type, grade I

Fig. 3—Squamous cell carcinoma, fungating type, grade III

Fig. 4—Peripheral fibrosarcoma

trating lesion (Fig. 1), a verrucous, superficially spreading type often associated with leukoplakia (Fig. 2), or a fungating lesion with an indurated base (Fig. 3).

Sarcomas are comparatively rare, and they also vary greatly according to histogenesis. The fibrosarcoma is the most common one and occurs as a peripheral tumor on the oral and cutaneous aspect of the jaws (Fig. 4). Occasionally it is seen as a central tumor (Fig. 5). The osteogenic sarcoma is an extremely rare tumor which may be osteolytic or osteoplastic or mixed, according to the degree of differentiation of the osteogenic cells and the presence of their by-products. Lymphosarcoma (Fig. 6), reticulum cell sarcoma and plasmocytomas (Fig. 7) and multiple myelomas are also uncommon in the mouth and jaws. When they do occur they are generally

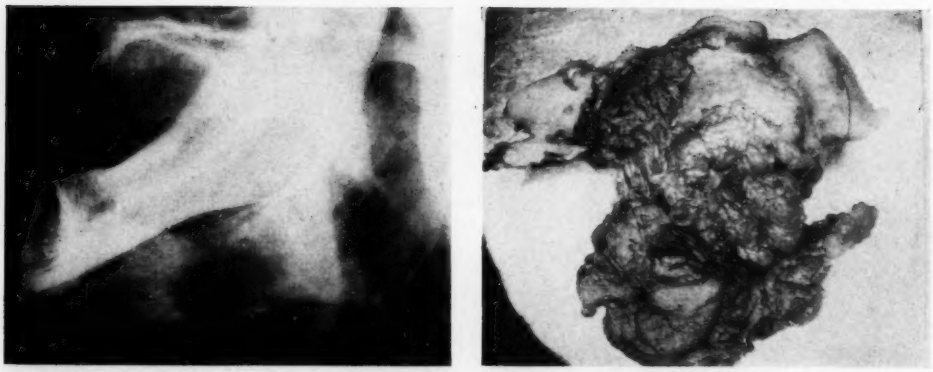

Fig. 5—Central fibrosarcoma. Left: Pathologic fracture. Right: Excision specimen

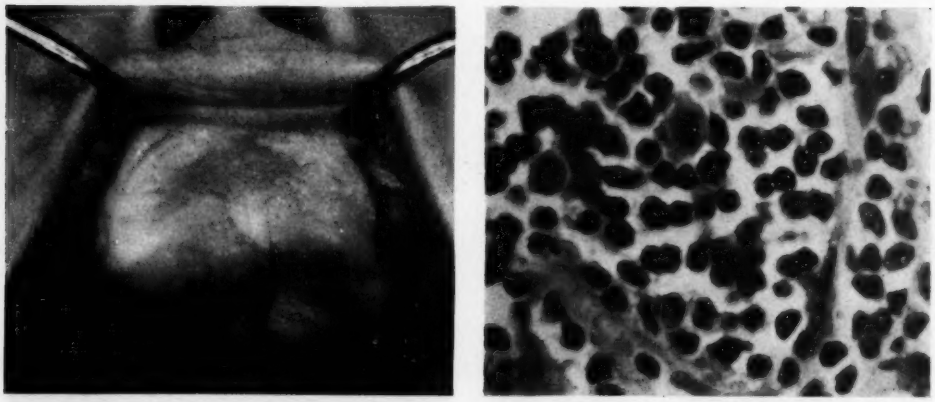

Fig. 6—Lymphosarcoma of maxilla. Left: Appearance of lesion. Right: Photomicrograph

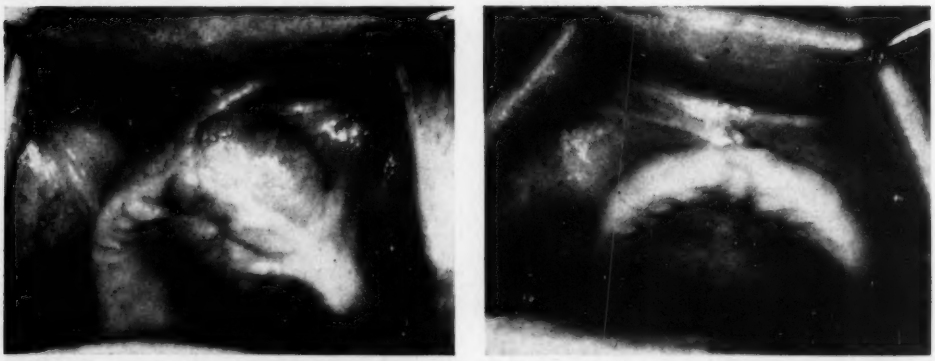

Fig. 7—Plasmocytoma of maxilla. Left: Before irradiation. Right: After irradiation

found in other parts of the body as well, being either of multiple origin or metastasizing early. A rare but very malignant tumor should finally be mentioned. This is the malignant melanoma which metastasizes to both lymph nodes and the vital organs very early (Fig. 8).

RAPIDITY OF GROWTH—The history of the lesion often will give a clue as to the rapidity of growth of a tumor, and the pathologist is able to determine the rapidity of cell multiplication by histologic examination. Rapidly growing tumors invade early and produce metastases while small. The invasion of blood vessels and lymphatics is a characteristic danger sign. As a rule, tumors in which the cells are predominantly embryonic in type are radiosensitive, whereas the presence of large numbers of differentiated cells is a sign of radioresistance.

LOCATION AND SIZE OF TUMOR—The location and size frequently governs the curability by surgery. It determines what type of resection is required to eradicate all disease completely.

In peripheral tumors it is important to note that a rapidly growing malignant tumor often spreads deeply into adjacent tissues without extending on the surface. Such lesions are more difficult to eradicate than a slowly growing, bulky tumor which does not invade the underlying structures extensively.

Central tumors generally are contained within the bone, but they may extend along the bony canals, in the lymphatics accompanying the nerves and vessels. In some instances the tumor may cause a perforation and then invade the surrounding structures rapidly.

Fig. 8—Malignant melanoma of maxilla. Above: Lesion. Below: Moore's incision used to excise maxilla

INVASION OF ADJACENT STRUCTURES—The invasion of fascial spaces or adjacent muscles must be considered, and if involved, electrosurgery may be necessary to make sure of complete eradication. This applies particularly to the floor of the mouth.

In the maxilla, the maxillary sinus may be involved. A careful roentgenographic study is indicated. If the lesion is far advanced, destruction of the bone is

Fig. 9—Squamous cell carcinoma, grade IV, invading maxillary sinus

characteristic, but this is not always seen in the early stages of invasion (Fig. 9).

In all peripheral tumors the bone may be involved. This is of considerable importance. The roentgenogram may be negative, in which case electrocoagulation of the surface of the bone adjacent to the lesion is carried out as a minimal procedure (Fig. 10). If grossly involved, and osteolytic changes are visible in the roentgenogram, resection of the involved part of the mandible is mandatory to prevent recurrence (Fig. 11).

When the tumor involves the mandibular canal, the mandible should be sectioned anterior to the mental, and posterior to the mandibular foramen, since the tumor may spread in the canal without roentgenographic evidence of it. In the maxilla, particular attention should be paid to the palatine canals, since extension of the tumor cells frequently takes place along the anterior palatine nerve and vessels (Fig. 12).

In central tumors of the jaws, pathologic fracture may be encountered. If this occurs, the surrounding soft tissue soon becomes invaded (Fig. 5, left). This is an indication for wide excision, a block resection of the mandible with surrounding structures attached (Fig. 5, right).

PRESENCE OF METASTASES—A very important part in the diagnosis of malig-

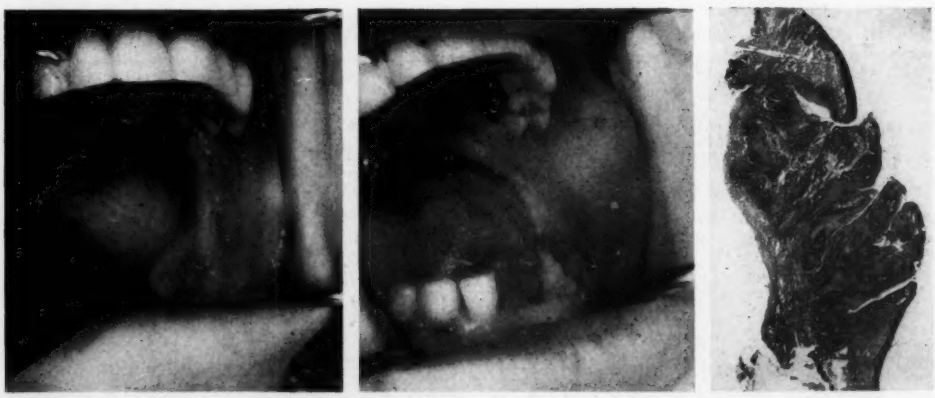

Fig. 10—Squamous cell carcinoma, grade II, of alveolar mucosa. Left: Lesion. Center: Area of necrotic coagulated bone. Right: Photomicrograph of lesion

nant disease is the determination as to whether the lesion is entirely local in nature or whether other organs are involved. It must be remembered that carcinoma metastasizes to the lymph nodes first. In invasive types of tumors the involvement of the regional nodes may occur very early, whereas in the slowly growing type, it may not occur even though the primary lesion is bulky and large. Sarcomas metastasize through hematogenous channels long before a carcinoma would. The malignant melanoma will spread along both the lymphatic and hematogenous paths. In all instances in which involvement of the vital organs is suspected, a careful physical examination including roentgenograms of the lungs is mandatory.

This leaves for consideration malignant lesions which are discovered first in the jaws but which are metastases from primary lesions somewhere else, or which are part of a multiple and simultaneous occurrence of tumors, as is the case in lymphoma, reticulo cell sarcoma and multiple myeloma. In such instances local excision is useless unless the disease can be controlled elsewhere. Sometimes metastases cannot be diagnosed positively. One patient (Fig. 8) had a melanoma of the right tuberosity. Examination revealed a suspicious lesion in the lung. The excision of the local lesion (Fig. 8, above) through Moore's incision (Fig. 8, below) was carried out successfully, but the lung lesion progressed to become an active metastasis which terminated fatally.

■ *Treatment of Malignant Tumors of the Jaws*

In planning treatment, the choice between surgery or radiation therapy must be made first. It is true that in many instances the primary lesion may be effectively controlled by irradiation. However, the complications which frequently arise after irradiation of bone produce deformi-

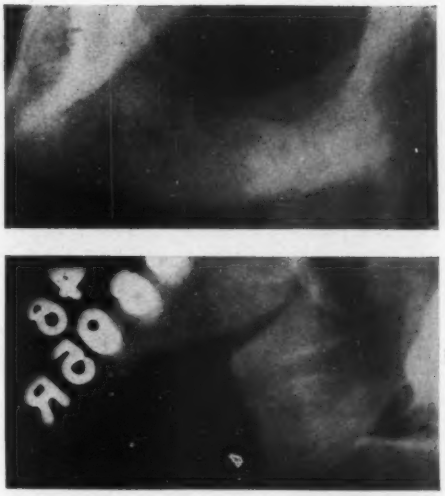

Fig. 11—Squamous cell carcinoma, grade II. Above: Roentgenogram showing invasion of mandible. Below: After resection of affected segment

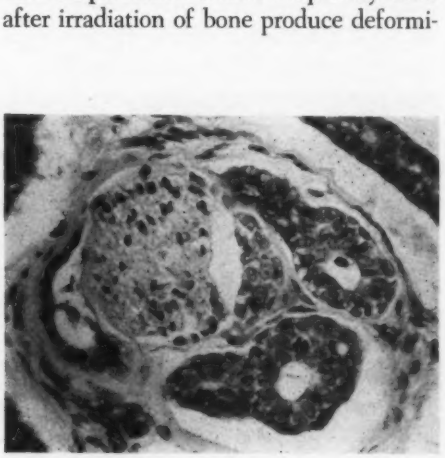

Fig. 12—Adenocarcinoma of hard palate showing invasion of lymphatics accompanying palatine nerve

Fig. 13—Irradiation necrosis of mandible in patient who had been treated with roentgen rays eight years before for extensive squamous cell carcinoma, grade I. Wearing of denture caused slough after cancer was cured

ties much more serious than those caused by radical surgery, with the additional handicap that the damaged tissue prevents reconstructive surgery such as may be achieved by the use of bone grafts. Even prostheses cannot be used in some instances because they may cause the breakdown of tissue that has been irradiated (Fig. 13). In addition, radiation therapy is generally ineffective in eradicating the secondary lymph node metastases in the neck.

Surgery, therefore, should be the treatment of choice unless contraindicated by the patient's general health. It should always be kept in mind that in cancer surgery, complete eradication at the first procedure is the aim. Secondary operations when there is recurrence seldom are successful.[1] The following methods are available:

LOCAL EXCISION OF THE PRIMARY LESION—Excision of the primary lesion will be successful in many instances if done early enough and if no metastases are present. The excision, however, must be liberal and carried out in the normal tissue surrounding the tumor (Fig. 14). In addition, the patient must be checked at regular intervals for local recurrence and the development of lymph node

1. Thoma, K. H. Oral surgery, ed. 2. St. Louis, C. V. Mosby Co., 1952.

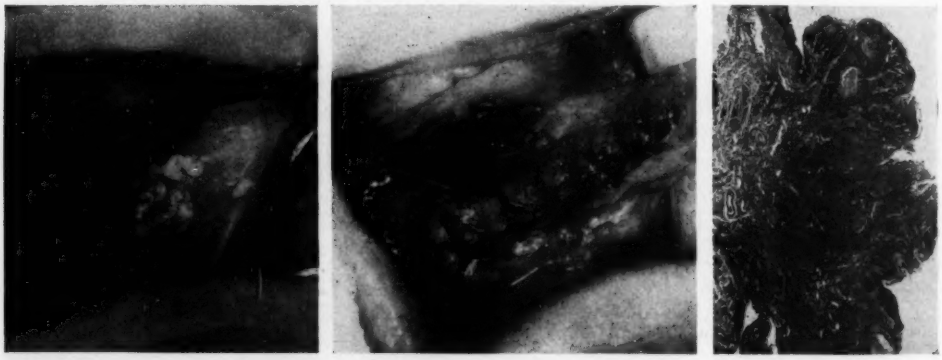

Fig. 14—Squamous cell carcinoma of tongue, grade II. Left: Lesion surrounded by leukoplakia. Center: Excision. Right: Photomicrograph showing papillomatous lesion with little invasion

metastases. It must be remembered that lymph nodes may not appear to be involved at the time of operation, but they may harbor a few tumor cells which cause progressive enlargement of the node later.

LOCAL EXCISION OF THE PRIMARY LESION WITH ELECTROCOAGULATION—If there is extensive soft tissue involvement in the floor of the mouth, or if the lesion occurs close to the bone, local excision of the primary lesion with electrocoagulation is the procedure used. The effect of electrocoagulation on the bone is localized necrosis (Fig. 10). Generally the affected cortex will sequestrate, and when the sequestrum is exfoliated epithelium will cover the defect.

SECTIONING OF MANDIBLE FOR RETRACTION, BLOCK EXCISION OF TUMOR AND REPAIR OF MANDIBLE—Sectioning of the mandible and retraction gives good exposure for excision of tumors of the tongue extending into the floor of the mouth. An excellent functional and cosmetic result is produced. After the excision, which is performed while the sectioned mandible is retracted laterally and which may include lymph node dissection in continuity, the mandible is wired together and the soft tissues closed by suture.

RESECTION OF AFFECTED BONE SEGMENT EN BLOC WITH LESION—The bone must always be sacrificed if it has been invaded by the lesion or if the submaxillary nodes attached are involved (Fig. 15). The resection again is an en bloc type of excision, with all involved tissue being removed attached to the jaw. In the maxilla, Moore's incision will greatly facilitate the procedure if the lesion is unilateral (Fig. 16). If the palatal carcinoma is extensive, the entire maxilla may have to be sacrificed (Fig. 17). If the lesion is in the mandible, the incision should be made 1.5 to 2 cm. beneath the inferior border of the jaw to prevent sectioning of filaments of the facial nerve and resultant facial paralysis.

RADICAL NECK DISSECTION—Radical neck dissection is indicated in all instances in which lymph nodes are involved by metastases. Prophylactic neck dissection may have a certain value and is advocated because involved nodes are not palpable at first. If the patient is watched closely at regular intervals, however, neck dissection may still effect a cure when indicated clinically. Today some surgeons recommend a one-stage, en bloc resection of the primary lesion including the field of lymphatic spread, whether lymph nodes are palpable or not. This type of surgery results in considerable cosmetic deformity, and since many instances of cancer have been cured by more conservative methods, a plea is made for the two-stage procedure.

The removal en bloc of the primary lesion by an oral surgeon who has made a specialty of tumor surgery and who is familiar with the resection and repair of oral structures is recommended. This applies especially in instances of early malignancy, the type of lesion the general dentist is able to recognize and refer for treatment. For the removal of the lymph nodes the patient should be referred promptly to the general surgeon, when indicated by clinical developments. The radical one-stage procedure should be reserved for instances of advanced cancer, and most of those who advocate it are general surgeons

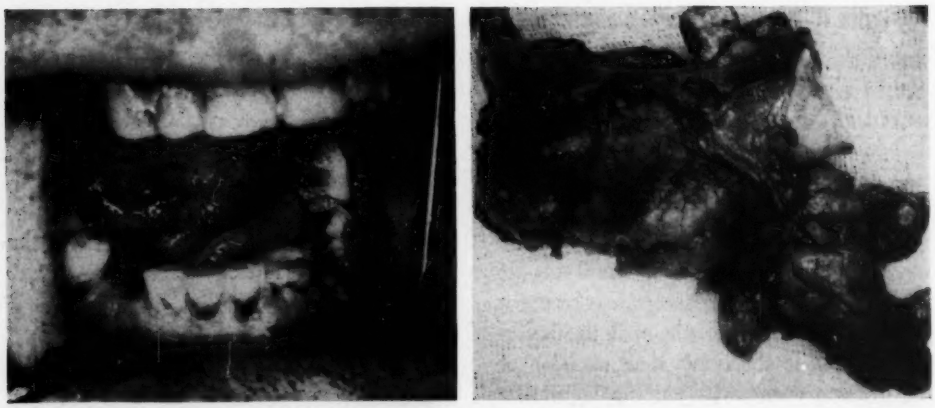

Fig. 15—Extensive squamous cell carcinoma, grade III. Left: Involvement of mandible, floor of mouth and ventral part of tongue. Right: Excision specimen—section of mandible with involved parts including submaxillary organs attached

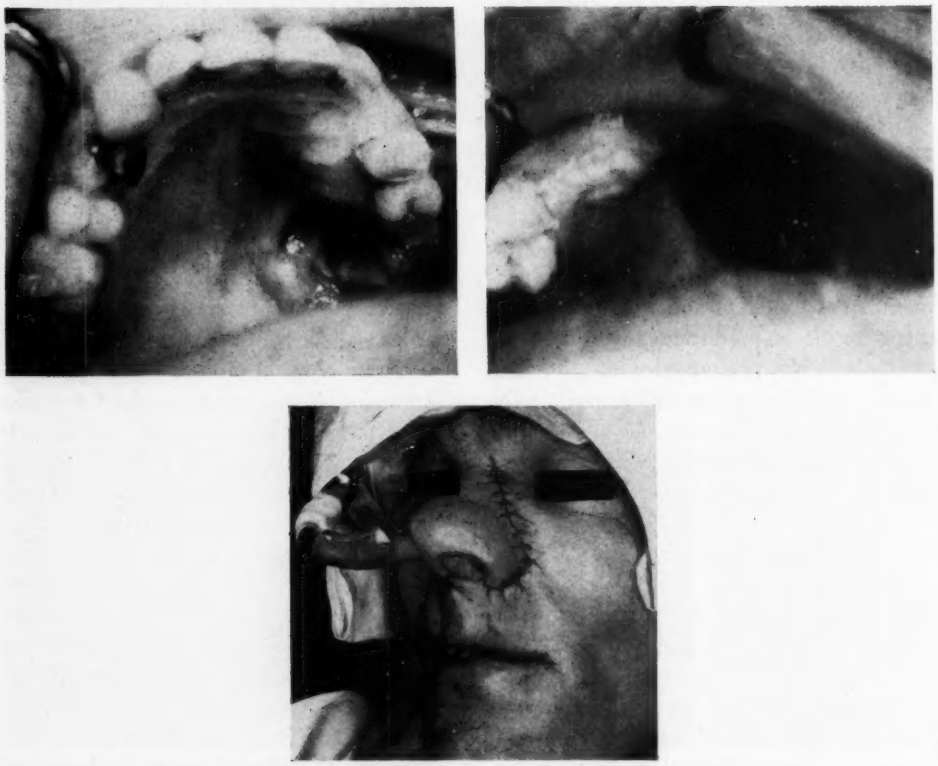

Fig. 16—Adenocarcinoma of maxilla. Left: Lesion. Center: Excision wound after healing. Below: Moore's incision used to excise left maxilla

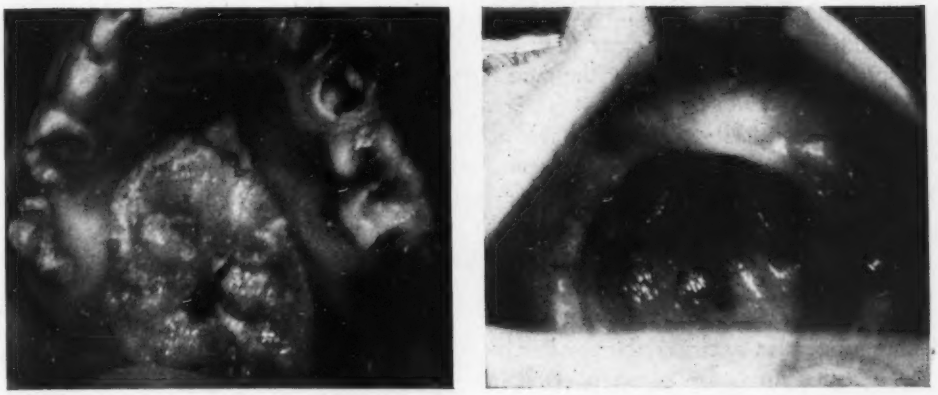

Fig. 17—Squamous cell carcinoma, grade III, of palate. Left: Perforation. Right: No recurrence one year after excision of maxilla

dealing with patients in whom the condition is far advanced who are seen more often in clinics than in private practice.

IRRADIATION—Although surgery has been given preference so far for the management of malignant disease, it should be pointed out that certain lesions are extremely radiosensitive so that their radiocurability is high, with the use of comparatively small doses.

The lymphosarcoma and reticulum cell sarcoma, also the plasmocytoma, come under this heading. It should be remembered, however, that some of these lesions are of multiple origin or metastasize early to other parts of the skeleton and, therefore, the patient is not necessarily cured if the oral lesion is controlled.

A lymposarcoma which developed on the palate of a woman, aged 61, is shown in Figure 6. The sinuses were normal but the chest roentgenogram showed a suspicious lesion in the hilus. The oral tumor receded completely after the administration of 3,200 r administered over a period of ten days. A woman, 61 years old, had a plasmocytoma of four to five weeks' duration in the anterior part of the palate (Fig. 10). The lesion involved the bone extensively but not the maxillary sinuses. Treatment was by irradiation. The administration of 1,900 r intraorally caused it to disappear (Fig. 7, right), and so far

Table

Advanced intraoral cancer in 137 patients Primary disease control and necrosis (From White, Sieniewicz and Christensen)

Tumor dose (r)	Number of patients	Per cent controlled	Per cent with necrosis
7,000- 8,999	33	36.3	12.1
7,000-10,999	71	49.3	18.3
9,000-10,999	38	60.5	23.8
9,000-12,000	50	51.0	28.0
7,000-12,000	83	48.2	21.7
Under 7,000	54	11.1	1.9

(one year) there has been no recurrence. In both instances, insufficient time has elapsed to determine whether the cure is permanent.

Squamous cell carcinomas should always be excised if possible. If the lesion is far advanced, however, and there is extensive involvement of various parts of the oral cavity, or if there are complications making surgery a great hazard, roentgen ray treatment may still effect a cure. Great improvement in roentgenotherapy has increased the number of instances that can be controlled, but large doses are required.

White, Sieniewicz, and Christensen[2] (1954) reported the results achieved with 137 patients who had advanced intraoral cancer treated at the Pondville Hospital. The results are shown in the table.

Even years after treatment, irradiation necrosis as can be seen by the analysis in the table, produces complications in patients in whom the cancer is cured as well as in those in whom it is uncontrolled. The immediate cause of the complication generally is extraction of a tooth or an erosion in the gingiva caused by the wearing of a denture which brings on infection of the necrotic bone and the surrounding tissue. Sloughing of the mucosa often exposes the entire alveolar portion of the bone, and submental or submaxillary abscesses may eventually develop. The condition resists antibiotic therapy and causes a great deal of suffering, and when part of the jaw has to be excised, produces a permanent deformity. An example is as follows:

The patient, 53 years of age, was first seen in 1948 with a squamous cell carcinoma, grade I, that extended over the floor of the mouth from the ventral part of the tongue to the anterior gingiva of the mandible without palpable nodes. After extraction of all the remaining teeth, the patient received roentgen ray treatment (12,000 r) in divided doses. His disease appeared completely controlled. In 1952 he had dentures made. These caused an erosion in the irradiated gingiva which slowly developed into an extensive slough that finally exposed the entire anterior part of the mandible (Fig. 13). In 1954 he developed a submental abscess and, in spite of antibiotic therapy and irrigations, a submental fistula remained which was discharging pus. Finally, five months later the necrotic part of the bone had to be excised approximately from the first molar region on the right to the first molar region on the left. Because of the effect of irradiation on his skin, no reconstructive surgery was possible, and he lives completely cured of cancer, but with an exceedingly receding chin.

■ *Conclusion*

The management of malignant disease in the oral cavity requires a great deal of experience. In no other type of disease is experience as important as in cancer treatment. A careful investigation should be made of the extent and nature of the lesion, and the type of therapy best suited for each individual instance should be considered carefully. The policy of consultation with other specialists involved in the management of cancer should be encouraged. The pathologist and the radiologist, in particular, can make valuable suggestions if the condition is unusual.—*1180 Beacon Street.*

2. White, G.; Sieniewicz, J., and Christensen, W. R. Improved control of advanced oral cancer with massive roentgen therapy. Radiology 63:37, 1954.

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CASE REPORTS

Severe allergic reaction during thiopental sodium anesthesia: *report of case*

■ *James R. Hayward,* D.D.S., M.S., Ann Arbor, Mich., and Gordon L. Kiester,† D.D.S., M.S., Orlando, Fla.*

Routine safe utilization of drugs may promote a false sense of security on the part of the surgeon and dull his alertness for the patient who may have the rare, but hazardous, combination of allergen and sensitivity response. The following case illustrates the necessity for precaution in the selection of anesthetic agents for patients with allergic tendencies.

A 23 year old dairy farmer was admitted to the hospital on March 3, 1955. His chief complaint was itching hives.

PAST HISTORY—The patient had been entirely well until January 1, 1955. At this time he developed urticaria beginning on his right hand, extending up his right arm the following week. The urticaria became migratory and transient in nature, involving the left arm, buttock and right ankle. The patient recalled that he had had two penicillin injections in the middle of December. He also recalled that he had had mild rhinorrhea and sneezing when he was in the barn.

His first hospital admission January 13 had revealed the following: generalized lymphadenopathy with large tender nodes in each axilla, multiple carious teeth, a periapical pathologic condition, and hives, 4 to 5 cm. in diameter, on the left elbow, right wrist, dorsum of the right foot, and left thigh.

The past history, systemic review and laboratory routines had been within normal limits except for a 10 per cent eosinophilia. The patient had been discharged on January 21, following a course of 50 mg. diphenhydramine hydrochloride four times a day every day and reserpine, 0.25 mg., twice a day every day. An axillary node biopsy while in the hospital had showed chronic nonspecific lymphadenitis.

PRESENT ILLNESS—The patient was readmitted March 3 for allergy checkup. The interim history was negative. Routines and physical examination revealed a normal white man with urticaria, dental caries and periodontitis. There were approximately 25 urticarial lesions measuring from 0.5 to 5 cm. in diameter scattered over his shoulders, trunk and thighs. He was placed on an allergy diet, avoiding the common allergenic foods. He was given no medication other than starch baths and Quotane lotion for local application to the lesions. While the patient was on this program for his first nine days in the hospital, the hives regressed slightly, but did not disappear.

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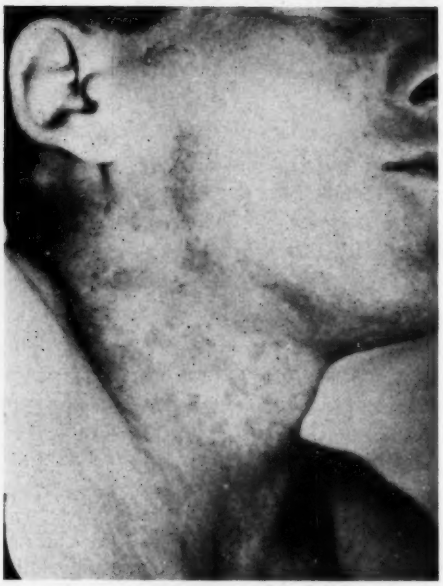

Urticaria manifestation of anesthetic drug reaction

The allergy consultant felt that the urticaria probably was due to the penicillin injections in December and that perhaps it was being perpetuated by penicillin in the cattle and poultry feed. Because of the possibility that infected teeth were acting as a focus of infection, full mouth extractions were scheduled using general anesthesia. No evaluation for drug sensitivity was conducted preoperatively.

TREATMENT—On March 11, the patient was premedicated with 100 mg. secobarbital sodium and 100 mg. meperidine hydrochloride. Atropine was omitted, but the anesthetist had planned to give 0.4 mg. as the intravenous anesthetic progressed. The anesthetic was 0.5 per cent intravenous thiopental sodium and was started at 7:55 A.M. By 8:03 A.M., 0.5 Gm. or 100 cc. had been given. Complete laryngospasm and massive urticaria developed. The urticaria consisted of huge elevated plaquelike hives (illustration). Intravenous administration of succinylcholine chloride was begun immediately. The spasm eased slightly, but partial laryngospasm lasted for eight minutes. Auscultation revealed widespread rales over both lungs. There were severe lid, periorbital and laryngeal edema. The patient perspired profusely. Further treatment of this spasm consisted of 50 mg. diphenhydramine hydrochloride (intravenous), 0.2 Gm. aminophylline (intravenous), hydrocortisone (intravenous), and 0.3 minims aqueous epinephrine (intramuscular) for hypotension. There was considerable clearing of the lung bases, and hives were diminished until 9:25 A.M., when they reappeared. Good results were obtained with the intravenous administration of 50 mg. diphenhydramine hydrochloride.

COURSE—The progress was satisfactory with the exception of shaking chills at 10:00 A.M. At 12:45 P.M. the patient's condition was aggravated considerably by an inadvertent intramuscular injection of pentobarbital. This was counteracted by speeding up the intravenous hydrocortisone acetate, and readministration of epinephrine, aminophylline, diphenhydramine hydrochloride and ACTH gel, 30 units intravenously. Recovery was slow since partial laryngospasm recurred, along with one moderately bloody brownish emesis. The rapidity of recovery from hives was also reduced greatly. The administration of ACTH gel, 30 units intramuscularly every 12 hours, cortisone 50 mg. four times a day every day and diphenhydramine hydrochloride, 50 mg. four times a day every day, gradually was tapered and finally discontinued on the fifth postoperative day. By the second day the hives completely disappeared.

Skin tests for procaine allergy were negative. On March 22, under local procaine anesthesia, full mouth extractions were performed without difficulty. Subsequent allergy skin tests were positive for mixed grasses, mixed weeds, indoor house dust, tobacco, wheat, celery, lettuce, white potatoes, gum tragacanth, buckwheat, penicillin and barbiturates. The patient was discharged on March 26, feeling well, with only mild residual lymphadenopathy.

SUMMARY AND CONCLUSIONS—A severe drug allergy reaction to thiopental sodium with anaphylactic shock and laryngospasm is reported. By exposing the obvious error in failing to respect the allergy status of the patient in selecting the anesthetic agent, it is hoped to stimulate oral surgeons to evaluate more carefully the ambulatory patients who require routine procedures under general anesthesia. Effective treatment for counteracting the anaphylactic reaction and laryngeal spasm has been illustrated in this report. The oral surgeon should be prepared to cope with this unusual situation.

Bilateral ostectomy for correction of prognathism: *report of case*

■ *Edward C. Armbrecht,* D.D.S.; George Clarke,† M.D., and Kenneth K. Kline,‡ D.D.S., Wheeling, W. Va.*

The case to be presented is of interest because it proves the psychological and functional value to a patient that is obtained by correction of a severe degree of prognathism of the mandible and illustrates the use of bone chips in the resection site.

The patient, a 30 year old, married, white, American woman was referred to the oral surgery service for treatment. Her chief complaints were inability to chew food satisfactorily and her feeling of looking grotesque to her family and friends (Fig. 1, left). She had noticed her inability to chew food satisfactorily after the posterior maxillary teeth had been extracted four years previously. After having been told by her dentist that dentures would be impossible to construct, she sought surgical correction of the deformity.

EXAMINATION—The patient was an obese, white woman with obvious prognathism of the mandible. The remaining upper teeth did not occlude with the scattered remaining lower teeth. Results of a routine physical examination were within normal limits.

PREOPERATIVE TREATMENT—Strict oral hygiene including oral prophylaxis was carried out. Impressions were taken of the upper and lower dental arches and poured into stone models and articulated. A silver splint was constructed on these models to be used as a postoperative splint (Fig. 1, above right).

After careful study of the articulated models, a two-stage ostectomy was decided on to correct the deformity.

FIRST STAGE OPERATION—The site of the ostectomy was marked out carefully and accurately on the stone models. The site of the resection was a location posterior to the lower right and left first bicuspids which involved also the second bicuspid and the first molar spaces on each side of the mandible. The space (7/16 inch) was marked off on the model. The first stage was performed under a bilateral mandibular block local anesthesia. The lower

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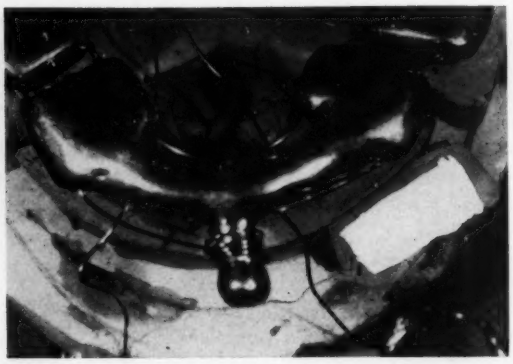

Fig. 1—Left: Preoperative profile of patient showing mandibular prognathism. Above right: Intraoral cast silver splint adapted to stone model

second bicuspids were extracted. The mucous membrane on the buccal and lingual surfaces of the bicuspid and molar space was elevated and the alveolar bone exposed on each side. The exact measurement, 7/16 of an inch, determined the bone drill cuts (posteriorly, anteriorly, and then transversely). The former two were parallel. The transverse cut was placed above the mandibular nerve canal, and the parallel cuts were extended below this level in the cortical bone plate so that later, in the second stage of the operation, the cuts could be identified easily and extended to the lower border of the mandible. The alveolar bone was removed *in toto* down to the transverse cuts. The mucous membrane was replaced and sutured. Postoperative reaction was minimal. The sutures were removed by the tenth postoperative day and healing was uneventful. A silver splint was cast, trimmed, and fitted to the model (Fig. 1, above right).

SECOND STAGE—Eight weeks later the patient was readmitted for the second stage of the operation. The usual preoperative preparations were carried out on the face, jaw and neck at the selected operative sites. Thiopental sodium, nitrous-oxide-oxygen endotracheal anesthesia was used for the operation. A skin incision was made below the inferior border of the mandible on each side, and dissection was extended down to the mandible. The periosteum was incised, dissected free and retracted. With the soft tissues retracted, the parallel oscillating circular saw blades were set at 7/16 of an inch, and the lower border of the mandible was cut to meet the former drill cuts made from within the oral cavity and near the lower surface of the mandibular canal. The bone between the cuts was chiseled out carefully to avoid injury to the mandibular nerve and vessels. Around the points, from which the nerve emerged in the posterior fragment and entered the anterior fragment, the bone was grooved so when apposition was secured there would be an adequate space for the excess of nerve and artery.

A 1/16 inch hole was drilled through each fragment, and a stainless steel .020 wire was passed through, tightened, and turned over so the wire ends would not irritate the tissues. The splint was then cemented to the teeth and secured with circumferential stainless steel wires (Fig. 2). The ostectomy regions were

Fig. 2—Postoperative result with cast silver splint and circumferential wires in place

saturated carefully with penicillin powder and were packed with numerous cancellous bone chips obtained from the resected specimen to fill in the minor defects that were present (Fig. 3). This was done to insure early bony union of the resected bone ends.

After the fragments were brought together, the chips were wedged into the groove of the separation line. The periosteum was then replaced over the mandible and sutured in place with catgut. The remaining layers were then closed in succession; the skin was closed with interrupted silk sutures and was dressed with gauze and collodion. A pressure bandage was applied.

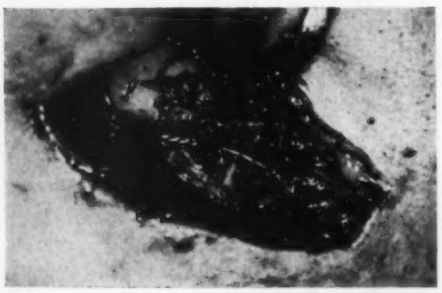

Fig. 3—Fragments reduced and immobilized with stainless steel wire. Bone chips can be seen in site of resection

POSTOPERATIVE TREATMENT—The postoperative course was uncomplicated. Feeding was handled through a polyethylene nasal gastric tube. Procaine penicillin G, 300,000 units, was given daily and 1 grain of phenobarbital was administered three times daily to control restlessness. Strict oral hygiene was maintained with hot saline mouth irrigation three to four times daily. The patient was discharged at the end of three weeks. Subsequently, the remaining upper teeth were removed under local anesthesia, and after primary healing had occurred, a temporary upper acrylic splint with six anterior teeth was inserted to maintain a normal vertical dimension while final healing of the ostectomy sites was taking place. Except for one episode of swelling at the left angle of the mandible, which was easily controlled by penicillin, recovery was uneventful (Fig. 4).

Fig. 4—Profile of patient after complete healing

COMMENT—Hullihen[1] in 1849 reported the first instance of surgical correction of mal-

relation of the jaws. Since then many oral surgeons have experimented with various technics. The procedure utilized in this instance was patterned after the Dingman[2] method. It varied somewhat in that the bone within the mouth was resected when the intraoral stage was done, thereby confining the final stage completely to the external surface and the lower border of the mandible, and cancellous bone chips were used in the resection area. At present, however, there is no accepted standard surgical technic that will fit all conditions and operators.[3]

CONCLUSION—Surgical technics for correction of mandibular prognathism have been described in medical and dental literature.[4,5] The two-stage method was selected as the one of choice for this particular instance. Bone chips placed in the resected areas enhanced the bone healing, and a cast silver splint held in place with circumferential wires provided excellent immobilization.

1. Hullihen, S. P. Case of elongation of the underjaw and distortion of face and neck, caused by a burn, successfully treated. Am. J.D. Sc. 9:157 Jan. 1849.

2. Dingman, R. O. Surgical correction of developmental deformities of the mandible. Plast. & Reconstr. Surg. 3:124 March 1948.

3. Gonzáles-Ulloa, Mario. Temporo-mandibular arthroplasty in the treatment of prognathism. Plast. & Reconstr. Surg. 8:136 Aug. 1951.

4. Thoma, K. H. Y-shaped osteotomy for correction of open bite in adults. Surg., Gynec. & Obst. 77:40 July 1943.

5. Waldron, C. W.; Karleen, C. I., and Waldron, C. A. Fundamentals in the surgical treatment of mandibular prognathism. Plast. & Reconstr. Surg. 4:163 March 1949.

Surgical correction of masseter muscle hypertrophy associated with unilateral prognathism: *report of case*

■ *Roy J. Eubanks, D.D.S., Dallas, Texas*

Masseteric hypertrophy is a rare condition in which one or both masseter muscles have within their substance an ill-defined contractile mass which gives an abnormal appearance to that area of the face. There have been only a few instances reported in the literature, and it is believed that none were in conjunction with a mandibular overgrowth, as was present in the case to be described.

The etiology of the condition is not clear but the "work hypertrophy" theory advanced by Thoma[1] seemed to apply in this instance because of the patient's inability to masticate equally on both sides of the mouth. Of significance also is a projection of cortical bone at the mandibular angle along the area of the muscle's insertion. This was a consistent finding in a series of cases reported by Masters, Georgiade and Pickrell.[2]

Surgical treatment of hypertrophy of the masseter muscle involves the resection of the hypertrophied musculature, as well as any ab-

1. Thoma, K. H. Oral pathology, ed. 3. St. Louis, C. V. Mosby Co., 1950, p. 1025.

2. Masters, F.; Georgiade, N., and Pickrell, K. Surgical treatment of benign masseteric hypertrophy. J. Plast. & Reconstr. Surg. 15:215 March 1955.

normal bony spur formation at the angle of the mandible. Cosmetic contouring of the muscle is preferably carried out on its medial surface, thereby minimizing the possibility of injury to facial nerve branches lying external to the muscle. In the following instance surgical resection of the muscle from its medial surface, with a prognathic correction as advocated by Smith,[3] were both accomplished through a single submandibular incision.

REPORT OF CASE—In June 1955, a 17 year old white boy was referred by his physician for diagnosis and treatment of a mandibular tumor. A general physical examination had revealed nothing abnormal other than a mass overlying the left mandibular ramus. The patient's chief concern was his facial disfigurement. He stated that several years prior to the examination he noticed that the left side of his face was larger than the right, and that it had become progressively more "lopsided." His parents concurred with the patient that for as long as they could remember he had chewed his food only on the left side.

EXAMINATION—Clinical examination revealed an ill-defined mass, apparently within the substance of the left masseter muscle (Fig. 1). It was visible and palpable in both the relaxed and contracted states of the muscle, becoming firmer and somewhat more circumscribed when the teeth were clinched. The right masseter muscle appeared to be somewhat underdeveloped.

On occluding the teeth the patient exhibited an end to end relation of the incisors, with a slight crossbite condition existing on the right side (Fig. 2).

Roentgenograms in the posteroanterior projection revealed inequality of bone growth at the right and left mandibular angles (Fig. 3). Lateral and temporomandibular roentgenograms showed both condyles to be in normal position within the glenoid fossa. A diagnosis of benign masseteric hypertrophy with left unilateral mandibular overgrowth was made.

TREATMENT—Maxillary and mandibular study models were made, duplicated and mounted on an articulator. By trial position and measurements, it was determined that 0.5 cm. posterior displacement of the mandible on the left side would bring the teeth into excellent occlusion. Surgical treatment planned was resec-

Fig. 1—Preoperative appearance

Fig. 2—Preoperative occlusion

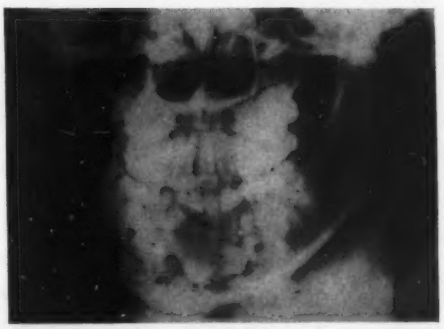

Fig. 3—Preoperative roentgenogram. Note projection of bone at left mandibular angle

3. Smith, A. E., and Robinson, M. Evaluation of physiologic result from submandibular-notch ostectomy, condylotomy operation for prognathism. J. Plast. & Reconstr. Surg. 15:197 March 1955.

tion of hypertrophied portion of the masseter muscle with subsigmoid notch ostectomy, sliding condylotomy through a submandibular incision.

The patient was hospitalized, and the surgery performed under thiopental sodium, nitrous oxide and oxygen nasal-endotracheal anesthesia with continuous intravenous infusion of Anectine chloride. After the patient was anesthetized, fracture splints were placed on the maxillary and mandibular teeth, and the patient was draped for the extraoral procedure.

A 6 cm. incision was made through the skin 1 cm. posterior and inferior to the lower border of the mandibular ramus. The platysma muscle was sectioned, and the anterior facial vein and facial artery cut between hemostats and tied. The incision was carefully deepened under strict hemostasis, and the marginal mandibular and buccal branches of the facial nerve identified and preserved.

The masseter muscle was separated from its insertion at the lower and posterior border of the ramus and elevated. A surgical contouring of the muscle was carried out on its medial surface until its thickness was reduced to normal proportions. The projection of cortical bone at the mandibular angle was next removed with chisels and rongeur forceps. The anesthetist was instructed to increase the Anectine chloride for maximum muscle relaxation, and the periosteum, masseter muscle and overlying tissues forcefully retracted to expose the sigmoid notch. Two vertical cuts, 0.5 cm. apart, were made through the ramus with a no. 558 cross-cut fissure bur from the sigmoid notch inferiorly for 0.8 cm.

A horizontal cut joined the inferior extensions of the previous cuts, thereby effecting ostectomy of the circumscribed area of bone. At this point two small drill holes were made, one through the proposed anteroinferior angle of the proximal fragment and the other in the ramus below the anterior vertical cut. An .018 stainless steel wire was threaded between the two holes. The horizontal cut was continued posteriorly to complete the condylotomy. The ramus was moved posteriorly into its newly created relation with the proximal fragment and was immobilized with the interosseous wire.

The drapes were rearranged so that the mouth could be reached by the assistant who placed the teeth in occlusion and maintained their position with intermaxillary elastics. After closely inspecting the surgically created fracture site for position and stability, the masseter muscle was sutured back to its bed on the ramus, and the overlying tissues closed in layers without drains. A pressure bandage was applied.

COURSE—During and after the surgery, the patient was given 2,000 cc. of fluid intravenously. He had mild postoperative edema and little sedation was required. The usual post-

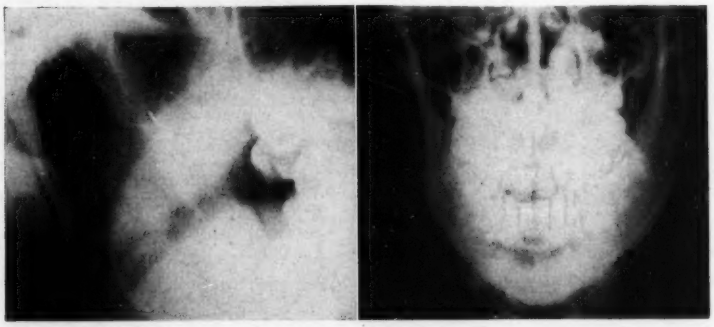

Fig. 4—Left: Postoperative roentgenogram showing new relation of ramus to condylar neck. Right: Postoperative view showing bone spur has been removed

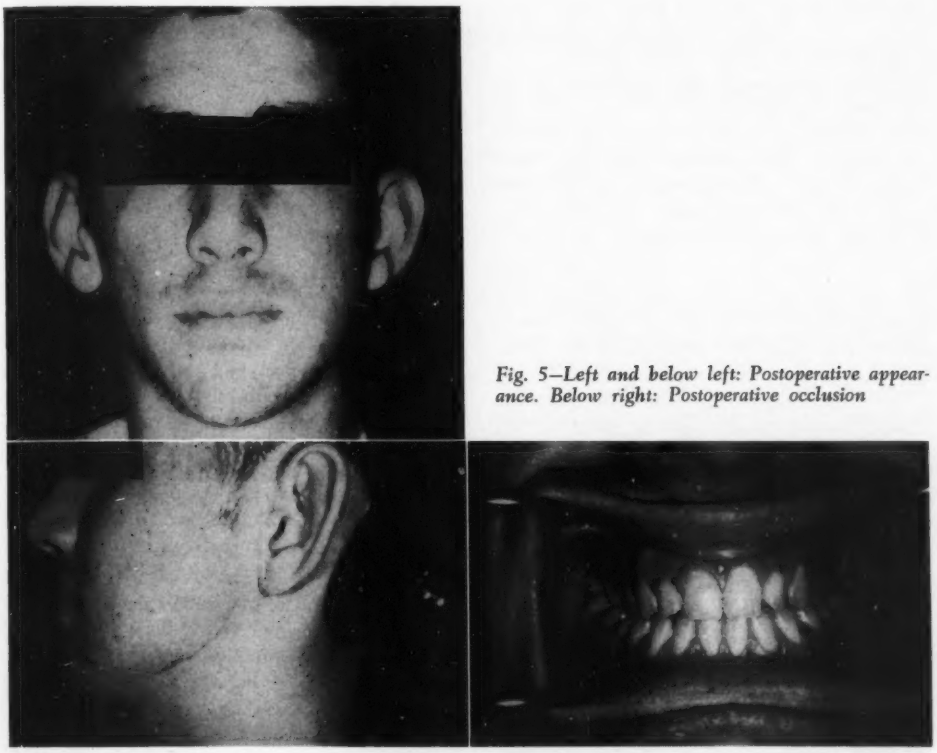

Fig. 5—Left and below left: Postoperative appearance. Below right: Postoperative occlusion

operative care given in prognathic operations was employed, including a special formula liquid feeding and antibiotic therapy.

The patient was discharged from the hospital on the fifth postoperative day, at which time the skin sutures were removed, and intermaxillary elastics changed to wire fixation. Roentgenograms made at this time showed the new relation of the condylar neck to the mandibular ramus (Fig. 4). Intermaxillary fixation was removed at the end of the seventh postoperative week, at which time the patient was instructed to masticate on the right side only, with the aim of developing the right masseter muscle.

Figure 5 shows the patient six months postoperatively, at which time his facial appearance was considerably improved and his occlusion corrected. Normal mandibular function was achieved.

SUMMARY—An unusual instance of facial asymmetry was corrected through surgical intervention. One submandibular skin incision afforded adequate access for both masseter muscle resection and for prognathic correction in the mandibular ramus. Pathologic examination of the muscle removed showed enlarged but normal striated muscle tissue. The bone spur was cortical bone.—223 *West Tenth Street.*

Muco-epidermoid tumor of the palate: *report of case*

■ *A. P. Chaudhry,* B.D.S. (India), M.S., Ph.D., and A. R. Stoesz,† D.D.S., Minneapolis*

Bhaskar[1] and co-workers have evaluated 181 instances of the minor salivary gland tumors that have thus far been described in the literature. An additional case report, presented herein, is of interest, not only because of the rarity of such tumors, but also from the point of view of its diagnosis and management.

REPORT OF CASE—A 30 year old white woman was seen at the University of Minnesota Dental School on January 10, 1956, with the chief complaint of a swelling in the palate. The mass had been noticed first in September 1954 at which time it was incised and drained. The swelling had gradually reappeared over a period of a year and had been quite noticeable in June 1955. At that time the left upper second molar had been extracted. There was no improvement, therefore, the patient consulted her dentist who referred her to the School of Dentistry for diagnosis and treatment.

At the time of admission to the oral diagnosis clinic, examination revealed a firm, nontender, nonfluctuant, 2 by 2 cm. swelling at the junction of the hard and soft palates to the left of the midline (Fig. 1). The swelling was not clearly demarcated, and there was no gross evidence of encapsulation. A small fistulous tract was seen and a mucinous material could be expressed on pressure from this tract.

Roentgenographic examination revealed a radiolucent defect more in keeping with bone resorption from pressure than invasion by neoplastic cells.

On the basis of the diffuse nature of the tumor, its location, and the presence of a fistulous tract exuding mucinous material, a presumptive diagnosis of muco-epidermoid tumor was made.

OPERATIVE PROCEDURE—On January 20, after adequate preoperative sedation, anesthesia of the left palate and tuberosity was obtained by local nerve block and infiltration of the soft tissues adjacent to the tumor. An incision was made from the tuberosity to the cuspid tooth on the same side, severing the lingual gingival attachments of the teeth. Elevation of this large mucoperiosteal flap brought the tumor mass into view.

There was no demonstrable capsule enveloping the mass which necessitated careful sharp dissection of abnormal from normal soft tissue. Resorption of palatal bone was evident; however, the tumor separated from the bony wall with ease, leaving a smooth clean surface. The anterior palatine vessels were visualized at their point of emergence from the greater palatine foramen and were clamped, cut, and ligated. The anterior palatine nerve was freed of tumor tissue and preserved intact.

Since no line of cleavage could be found between tumor and oral mucosa, a small section of mucosa was excised along with the tumor mass. Cauterization of the cavity was accomplished with 90 per cent phenol, followed by neutralization with absolute alcohol. The flap was returned and held in place by means of multiple no. 000 silk sutures. An antiseptic, analgesic gauze strip dressing was placed into the cavity through the mucosal defect, the cavity being judged too large to permit primary closure

*Research fellow in oral pathology, School of Dentistry, University of Minnesota.

†Resident in oral surgery, School of Dentistry, University of Minnesota.

1. Bhaskar, S. N., and Weinmann, J. P. Tumors of the minor salivary glands. Oral Surg., Oral Med. and Oral Path. 8:1278 Dec. 1955.

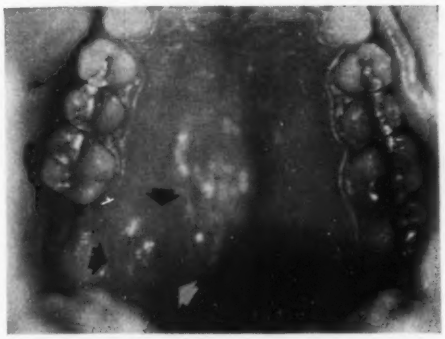

Fig. 1—Muco-epidermoid tumor of the palate (mirror view, sides reversed)

without undue tissue tension. Adequate prophylaxis against postoperative infection was provided by intramuscular injection of 600,000 units of long-acting benzathine penicillin G.

Examination of the patient on the fourth day after the operation revealed an uneventful postoperative course. The cavity was irrigated and a fresh gauze dressing inserted. All sutures were left in place.

The patient was again seen on the seventh postoperative day, at which time all sutures were removed and the gauze dressing changed. The entire flap was viable and had good color. Granulation tissue covered the walls of the cavity.

Dressing of the wound was discontinued during the third week after surgery. Examination of the patient five weeks postoperatively showed the cavity to be filling rapidly with healthy granulation tissue. Two and one-half months after surgery the cavity was completely obliterated and covered by normal epithelium. There was no clinical evidence of tumor recurrence.

PATHOLOGIC FINDINGS—The pathological report was as follows: "The tumor tissue is primarily made up of enlarged and disorganized acini and ducts. Some of these are lined peripherally with columnar epithelium and their lumina are filled with mucin, mucin-producing cells and epidermoid cells. At places the glandular pattern is distorted, being replaced by several layers of epidermoid and mucin producing cells. There are only a few scattered areas of chronic inflammation with no evidence of foreign body giant cell reaction. Diagnosis: muco-epidermoid tumor (benign variety)" (Fig. 2).

DISCUSSION — Muco-epidermoid tumors, both the benign and the malignant variety, were recognized as a separate clinical entity by Stewart, Foote and Becker.[2] The muco-epider-

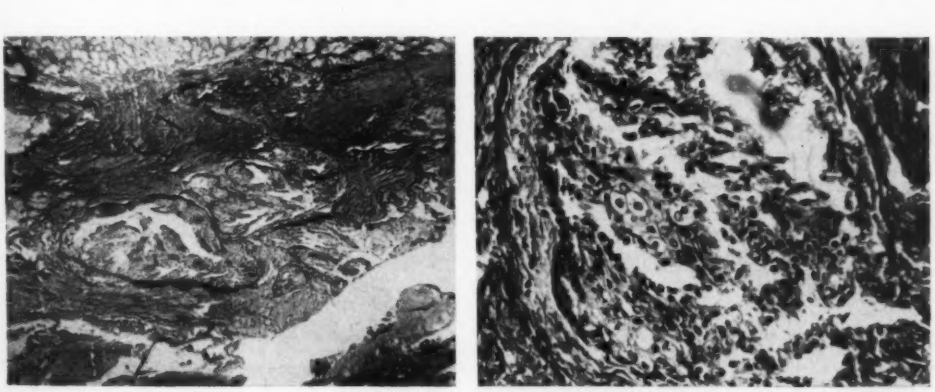

Fig. 2—Left: Photomicrograph of benign muco-epidermoid tumor showing ducts filled with epidermoid and mucin-producing cells. Right: Higher magnification

moid tumor is one of the most common tumors of the minor salivary glands. Smith[3] and co-workers reported that 30 per cent of the minor salivary gland tumors are muco-epidermoid. Bhaskar[1] and co-workers found 26 per cent of their 23 instances belonged to this variety.

Stewart, Foote and Becker, from extensive study of this neoplasm, concluded that it is not always easy to distinguish the benign from the malignant varieties, and that the former type often recurs and the recurrences may be malignant. Bernier[4] subscribes to their viewpoint and suggests that benign tumors occur at an earlier age, are less aggressive, and secrete abundant mucin, whereas the malignant variety exhibits rapid growth and excessive proliferation with formation of sheets of small, hyperchromatic, rounded cells. Most pathologists suggest that muco-epidermoid tumors be considered malignant, or at least as potentially malignant, irrespective of their histologic structure. Accordingly, surgical eradication of such tumors seems to be desirable.

2. Stewart, F. W.; Foote, F. W., and Becker, W. F. Muco-epidermoid tumors of salivary glands. Ann. Surg. 122:820 Nov. 1945.
3. Smith, A. G.; Broadbent, T. R., and Zavaleta, A. A. Tumors of oral mucous glands. Cancer 7:224 March 1954.
4. Bernier, J. L. The management of oral diseases, ed. 1. St. Louis, C. V. Mosby Co., 1955, p. 601.

Bilateral coronoid process exostoses simulating partial ankylosis of the temporomandibular joint: *report of case*

■ *Wilbur N. Van Zile,* D.D.S., St. Albans, N. Y., and William B. Johnson,† D.D.S., Great Lakes, Ill.*

Bilateral exostoses of the mandibular coronoid processes of sufficient magnitude to impinge against the zygomas and simulate a bilateral partial temporomandibular joint ankylosis have not been reported in medico-dental literature so far as could be determined. The case described herein presented an unusual diagnostic problem. The results of treatment were extremely gratifying.

REPORT OF A CASE—A 30 year old white man was referred to the oral surgery service of a naval hospital for consultation because of his inability to open his jaws adequately. After preliminary examination, it was recommended that he be hospitalized for further study and evaluation. He was admitted on December 11, 1953.

COMPLAINT AND HISTORY OF PRESENT ILLNESS—The patient's chief complaint, that of inability to open his jaws, was further elaborated to include pain in both ears during deep aircraft dives and an inability to manage

The opinions or assertions contained herein are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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Fig. 1—Maximum opening before treatment measured between the incisal edges of the anterior teeth was 7 mm.

an oxygen apparatus properly under flying conditions.

The aural pain, while in flight, was accompanied by impaired hearing which could be relieved only by vigorously blowing the nose to inflate the eustachian tubes. At the age of 14 he had been aware of an abnormal restriction in opening his mouth which had become progressively more restricted over the years.

In 1945 he had been hospitalized for consideration of his complaint. He had been told that roentgenograms revealed no abnormalities or significant findings in relation to his complaint, and no treatment was instituted. In January 1953, when he had been seen by an oral surgeon on a subsequent tour of military duty, both temporomandibular joints were injected with a solution, the name of which he did not know, nor was this drug recorded in his medical record. In April 1953 and again in June 1953, he had been hospitalized for the same complaint. In both instances a diagnosis of partial fibrous ankylosis of the temporomandibular joints was made and the patient told that no treatment was indicated.

PAST HISTORY—The patient's past history was negative and unrevealing with regard to his chief complaint. He had experienced no acute or chronic illnesses and had never suffered traumatic facial injury.

ORAL AND ROENTGENOGRAPHIC EXAMINATIONS—Oral examination, which was difficult because of the small interdental space, revealed a normal dentition with normal centric relationship. The oral tissues were healthy in appearance except for the presence of incipient gingivitis. Dental restorations were in poor repair. (The inability of the patient to open his mouth had precluded dental treatment for several years.) The lateral excursive movements of the mandible were present but very restricted. At maximal forced opening, a 7 mm. freeway space was measured between the incisal edges of the maxillary and mandibular teeth (Fig. 1), and the restrictive force felt firm and solid rather than resilient. In this position the mandible was seen to deviate slightly toward the right, and lateral movements of the mandible were not possible. Palpation of the mandibular condyles in the external auditory canal revealed little movement when the patient opened his mouth, and the tips of the coronoid processes could not be palpated intraorally in the open position. There was no evidence of asymmetry or depression of the zygomatic arches, and the patient's face was of normal contour.

Roentgenograms of the temporomandibular joints in the open and closed position were reported by the roentgenologist as normal, except for lack of excursive movement, and negative for evidence of pathologic change. In view of the patient's past history, which seemed to present no logical systemic or traumatic etiologic factor to account for temporomandibular joint disease or derangement, consideration was given to the possibility of anatomic abnormality of the coronoid processes. Careful study of the temporomandibular joint roentgenograms suggested a deformity of both coronoid processes (Fig. 2). Stereoscopic Water's roentgenograms of the open position were then ordered, which revealed abnormality in mediolateral thickness of the coronoid processes and apparent contact between the coronoid processes and the anteromedial surfaces of the zygomatic arches. Irregular, nodular radiopacities were also noted on both zygomatic arches at the point of impact (Fig. 3).

GENERAL PHYSICAL FINDINGS—Review of the systems and laboratory examinations were all reported as normal or negative in relation to the patient's complaint. Roentgenograms of the chest revealed no abnormality of heart or lung

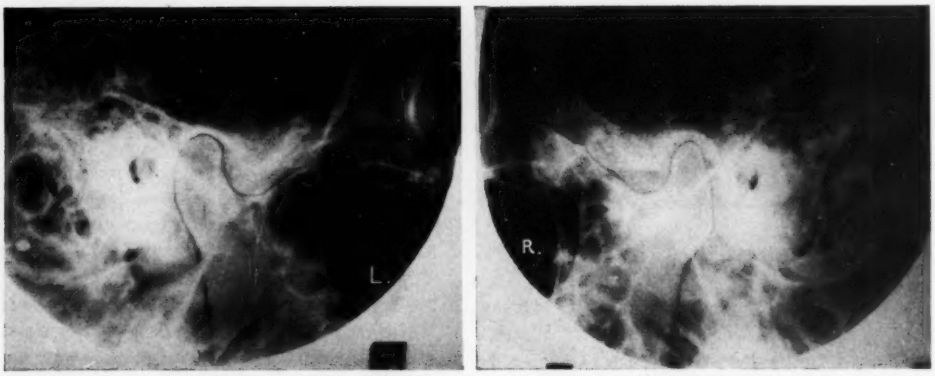

Fig. 2—Lateral roentgenograms of the hypertrophied coronoid processes (retouched for photographic clarity). Note gross elongation

fields. Blood studies were within normal limits and the results of the Kahn test were negative. The patient's blood pressure was 138/88, temperature 98.6° F., and pulse 92.

TREATMENT AND COURSE—A diagnosis of bilateral exostosis of the coronoid processes of the mandible was made on the basis of roentgenographic visualization of the point of impact between the coronoid processes and the zygomatic arches (Fig. 3) and the abnormality of anatomic form of the coronoid processes visualized in the lateral temporomandibular joint views (Fig. 2). The diagnosis was explained to the patient and a recommendation made for surgical excision of the abnormal portion of both coronoid processes. He readily agreed to the suggested surgical intervention. On January 21, 1954, the patient was prepared for surgery on the following day.

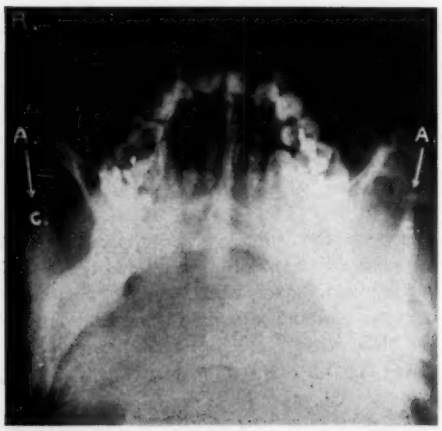

Fig. 3—Anteroposterior roentgenogram. A, point of impact between coronoid process and zygoma; C, hypertrophied coronoid process. Note exostoses on the zygoma at the point of impact

On January 22, under general anesthesia consisting of intravenous thiopental sodium plus nitrous oxide-oxygen and ether administered through an endotracheal tube, a 4 cm. straight line incision was made just superior to the right zygomatic arch and carried to the level of the deep fascia. A diagonal incision was then carefully made through the deep fascia, paralleling the expected direction routes of branches of the facial nerve. The incision was carried through the periosteum of the zygoma, which was elevated and retracted anteroposteriorly.

Fibers of the masseter muscle below the zygoma were then penetrated with a small hemostat and separated by blunt dissection; thus the fibers of the temporalis muscle and the coronoid process were exposed. The point of impact between the coronoid process and the

medial surface of the zygomatic arch was located without difficulty. The hypertrophied tip of the coronoid process was sectioned with a surgical bur, the tendinous attachments of the temporalis muscle were cut, and the fragment was removed.

Sections of bone were also excised from the osseous formation on the medial surface of the zygomatic arch. The masseter and temporalis muscles were normal in appearance and without fibrosis. After completion of the dissection, the deep tissues were closed by anatomic layers with no. 000 plain gut interrupted sutures, and the skin was closed with an interrupted no. 000000 Dermalon suture.

The patient recovered uneventfully. On January 29, one week after coronoidectomy on the right side, the left coronoid process was excised in an identical manner. Before surgical intervention on the left side, the freeway space between the incisor teeth in the open position was 10 mm., which indicated an increase in the opening of 3 mm. as compared with the original 7 mm. opening. Immediately after completing the resection of the left coronoid process, the space between the incisal edges of the incisor teeth was measured at 22 mm. Four days later, physical therapy, which consisted of deep diathermy and functional exercises, was instituted.

PATHOLOGY REPORT—The pathologist's report was as follows:

"Gross Examination—The specimens consist of: (a) several fragments of bone taken from an 'osseous growth' on the right zygoma; and (b) two larger fragments of bone described as the 'tips of the right and left coronoid processes,' which appear to be considerably altered and grossly enlarged (Fig. 4). The right coronoid, which measures 20 mm. in length by 10.5 mm. in thickness, presents a roughened 'moth-eaten' appearance on the anterior surface. The left coronoid, which measures 21 mm. in length by 5 mm. in thickness, presents a roughened anterior surface near the tip.

"Microscopic Examination—All sections show thickened cortical bone beneath which the bone trabeculae are viable, the marrow spaces containing a mixture of red marrow and fat. On the anterior surface of the right coronoid section, the bone spicules are undergoing degenerative changes and the interstices between the bone trabeculae contain necrotic debris. There is no evidence of malignancy in any of the sections. Diagnosis—Benign exostoses."

Fig. 4—Excised coronoid processes (anterior surfaces). The points of impact with the zygomatic arches are indicated at A

RESULTS—At the time of discharge on February 18, 1954, the patient was able to eat normally for the first time in several years. The opening between the incisor teeth, which measured 27 mm. as compared with the original 7 mm. before surgical intervention, was progressively increasing with exercise and use (Fig. 5). The lateral excursions of the mandible, although restricted, were much greater than had been possible preoperatively. There was no evidence of facial nerve paralysis and facial scarring was minimal (Fig. 6).

Fig. 5—The patient's opening three weeks after bilateral coronoidectomy measured 27 mm.

Fig. 6—The surgical approach was planned for optimal cosmetic effect (three weeks after operation)

COMMENT—The etiology of the developmental deformity of the coronoid processes in this instance is obscure. Similar but not identical lesions of the coronoid process have been reported as osteochondroma. Thoma regarded osteochondroma of the jaws as a rare tumor.[1] There have been only five osteochondromas of the coronoid process reported in medico-dental literature, to the best knowledge of the writers, and all have been unilateral in occurrence.[2-5] In all the instances reported the observations have been similar, namely: (1) unilateral occurrence (which follows normal expectancy in so rare a tumor); (2) a "mushroom-shaped" tumor mass; and (3) osteolysis of the zygoma opposite the point of impact with the tumor mass. In the case described herein, the deformity occurred bilaterally, the deformities were not "mushroom-shaped" as is frequently true in osteochondroma, and there was osseous hyperplasia present on both zygomas at the point of impingement by the coronoid processes.

Osteochondromas are benign neoplasms which develop most frequently in patients between 10 and 25 years of age. A striking feature is that they are nearly always located in periarticular areas or are related to tendinous attachments to bone. Generally these tendons are involved in maximal traction and attach directly to bone rather than to periosteum.

HISTOGENESIS OF OSTEOCHONDROMA—According to Geschickter and Copeland,[6] osteochondromas are an exaggeration of a normal bony protuberance required for the anchoring of an important tendon. They stated: "In normal tendons which attach directly to bone . . . proliferating centers may be found. In these proliferating centers early cartilage cells derived from young fibroblasts may be seen. . . . The occurrence of such proliferating zones in normal tendon ends was first observed by Kolliker in 1853. These tendons have their osseous ends formed not by ordinary fibrous tissues, but by extraskeletal blastema and since the periosteum is normally lacking or deficient at such points, the underlying bone lacks a limiting membrane and grows out in the form of a normal bony protuberance to meet the tendon. . . . Therefore, the normal structures at the site of future exostoses, which must be superseded or distorted for tumor formation to take place, are: (1) The end of a tendon or ligament formed from blastemal tissue and destined to aid in the formation of its own bony attachments; (2) an underlying zone of normal bone without periosteal covering destined to form an outgrowth or attachment point in the skeleton; and (3) a margin of periosteum surrounding the zone which eventually is to form a sleeve or cuff about this zone.

"The origin of an osteochondroma or exostosis at such transitional zones depends on abnormal variations in some one of the foregoing factors. If both the zone of cartilage in the tendon end and the bony outgrowth of the

1. Thoma, K. H. Oral pathology. St. Louis, C. V. Mosby Co., 1950.

2. Brailsford, J. F. An unusual osteochondroma from the coronoid process of the mandible. Brit. J. Radiol. 25:555 Oct. 1952.

3. Shackelford, R. T., and Brown, W. H. Osteochondroma of the coronoid process of the mandible. Surg., Gynec. & Obst. 77:51 July 1943.

4. Shackelford, R. T., and Brown, W. H. Restricted jaw motion due to osteochondroma of the coronoid process. J. Bone & Joint Surg. 31A:107 Jan. 1949.

5. Brandt, K. Deformation congenitale de l'apophyse coronoid du maxillaire inferieur. Acta orthopaedica Scandinav. 14:219, 1943.

6. Geschickter, C. F., and Copeland, M. M. Tumor of bone. Philadelphia, J. B. Lippincott Co., 1949, ch. 2.

underlying bone are normally balanced and eventually overlaid by periosteum, an osteochondroma will not form. If, however, the periosteum surmounts only the outgrowth of the underlying bone and fails to blend with the fibrous tissue of the tendon, the growth of the cartilaginous center in the fibrous attachment of the muscle will not be properly limited and a tumor results. In such an event, the cartilaginous zone in the tendon overgrows the protuberance of normal bone, overlapping the periosteum and forming a typical exostosis."

CONCLUSION—The exostoses in the instance described occurred during the age period (10 to 25 years) when osteochondromas most frequently develop, as evidenced by the patient's knowledge of a restriction in ability to open his jaws when he was 14 years old. The site of the tumors presents all of the histogenetic requirements for the development of an osteochondroma, and it is therefore probable that the original tumors were osteochondromas which had reached maturity or maximum growth many years previously. With the continued chronic irritation at the point of impact, however, a superimposed reactive osseous hyperplasia developed bilaterally on both the coronoid process and the zygoma, thus continuously reducing the space over which the coronoid processes could move.

SUMMARY—A patient came for treatment of a condition in which bilateral exostoses of the mandibular coronoid processes had resulted in impaction with the zygomas, thus simulating a constrictive ankylosis of the temporomandibular joint. The lesions probably originated as osteochondromas and had reached maturity during the normal growth period after which a chronic reactive osseous hyperplasia was superimposed at the point of mechanical impact between the coronoid processes and the zygomas. Normal mandibular function was restored by surgical excision of both coronoid processes.

Five instances of unilateral osteochondroma of the mandibular coronoid process have been reported in medico-dental literature. The case history presented here is, to the best knowledge of the authors, the first instances of bilateral osteochondroma (exostoses) of the mandibular coronoid processes to be reported.

■ SCIENCE AND CIVILIZATION

Science is the great instrument of social change, all the greater because its object is not change but knowledge, and its silent appropriation of this dominant function, amid the din of political and religious strife, is the most vital of all the revolutions which have marked the development of modern civilization.—*A. J. Balfour, Decadence, 1908.*

Intraoral bone graft: *report of case*

■ *Harold G. Green, D.D.S., Camp Pendleton, Calif.*

Among the many oral surgical procedures that have improved in recent years is the use of graft tissues to eliminate mandibular defects. This method has resulted in shorter healing time, better contoured supporting structures and less discomfort and inconvenience to the patient. The following is an interesting case in which the intraoral approach was used for the autogenous bone grafting of a mandibular cavity.

HISTORY—The patient, a 24 year old Marine sergeant, was referred to the Naval Hospital for evaluation and treatment of a large mandibular cyst. His past history, family history and the review of his system were noncontributory with reference to his present illness.

PHYSICAL EXAMINATION—Physical examination revealed a well nourished, well developed, Caucasian man who was alert and cooperative. His temperature was 98°F., pulse 80, blood pressure 110/58. His head and neck in general were normal. There were no palpable lymph nodes in the facial or cervical area, and the mouth was normal except for missing mandibular teeth and the drifting of the anterior mandibular teeth to the right of the midline. The remainder of the physical examination was not remarkable.

ROENTGENOGRAPHIC AND LABORATORY REPORTS—The results of routine laboratory studies, including serology and urinalysis, were within normal limits. Examination of the chest revealed it to be normal. Preoperative examination of the mandible showed a large (2 by 3 by 2 cm.) cyst in the anterior portion of the mandible (Fig. 1). It appeared to be well encapsulated and contained amorphous, partially calcified material. There was no evidence of malignancy. Located below the left lower first molar was a retained bicuspid in a vertical position, whose apex rested in the mandibular cortex. On the right, and outside the cyst, was a similarly positioned bicuspid.

HOSPITAL COURSE—A diagnosis was made of odontogenic tumor and retained teeth. The patient was admitted to the ward and prepared for surgery. Preoperatively he was started on oxytetracycline, 250 mg. four times a day, and continued on that therapy for 25 days.

With routine preparation and under general anesthesia, removal of the tumor and retained teeth and placement of a bone graft was performed in the following manner: A mucoperiosteal flap was retracted in the buccal vestibule from the lower left first bicuspid to the lower right second molar. Seven teeth were removed. With osteotomes the labial bone was removed to expose a bluish cystic sac which extended to the lower border of the mandible and from cuspid to cuspid. It contained a calcified mass of dental material. Enucleation of the cyst and its contents revealed a clean, eburnated cavity. Two retained teeth located near the inferior mandibular border were sectioned and removed. While the tumor and the retained teeth were being removed, an iliac crest bone graft had been taken from the left ilium by the orthopedic surgeon. This was broken up and placed in a solution of penicillin and streptomycin. The iliac crest bone graft, in small pieces, was then placed in the cavities (Fig. 2). The mucoperiosteal flap was replaced and sutured. A prefabricated acrylic splint was then placed over the wound and fastened to the remaining posterior teeth.

On the third postsurgical day, swelling had reached its maximum and thereafter gradually subsided. Healing was uneventful. The histopathologic report was as follows:

Fig. 1—Preoperative roentgenograms showing large odontogenic tumor and retained teeth in anterior portion of the mandible

MACROSCOPIC EXAMINATION—The specimen was an irregular 3 by 1 by 2 cm. mass having an incomplete cyst wall composed of soft fibrous tissue mixed with hemorrhagic zones. Within these cysts was a large irregular calcified mass presenting a coarse papillary surface with several definite poorly formed tooth structures. Sectioning through the calcified zone revealed imperfect root formation embedded in extremely hard and calcified tissues.

MICROSCOPIC EXAMINATION—The sections from the calcified portion revealed large masses of dentin which were arranged in caricatures of teeth. Several separate formations were present. Interspersed between these zones were large masses of cementum. There was adjacent fibrous tissue containing nodules of cementum and remnants of the enamel organ. In some zones these remnants showed definite proliferation of the epithelial elements with early differentiation of the basal layer into structures resembling ameloblasts. The diagnosis was composite compound odontoma.

The evaluation made at the time of the patient's discharge indicated normal healing of the oral tissues. All swelling had subsided, and the temporary splint was giving good service. Examination six months later revealed a well healed and well formed alveolar ridge supporting a partial denture. Roentgenograms (Fig. 3) revealed good consolidation of the bone graft.

COMMENT—A 24 year old Marine sergeant came for treatment of a composite compound odontoma. Graft tissue was utilized to eliminate a mandibular defect and this resulted in a short, uneventful recovery. Points of interest in treatment of this case were as follows:

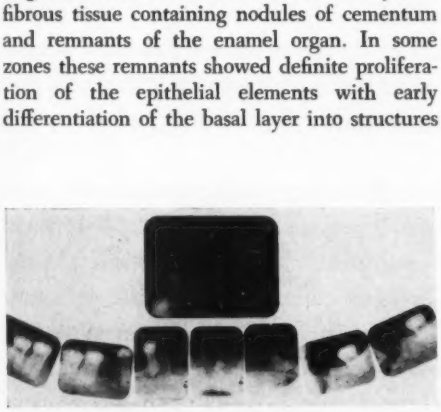

Fig. 2—Immediate postoperative roentgenograms showing mandibular cavity filled with autogenous bone chips

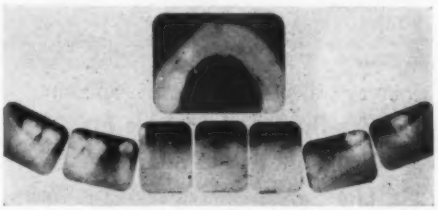

Fig. 3—Postoperative views taken six months later showing good consolidation of bone graft

1. The type and character of the bone used was autogenous, cortical and cancellous iliac bone chips which were utilized to provide a matrix for generation of new bone.

2. No attempt was made to regulate the vascular supply; however, it was supposed that sufficient blood and intercellular fluid was available for cellular nutrition on this level.

3. The mandibular periosteum covered a portion of the graft tissue.

4. The patient was young and healthy; the graft was placed intraorally; antibiotic therapy was utilized and no infection developed.

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Research in oral surgery

THE DEVELOPMENT OF additional, well-conceived research programs in oral surgery is urgently needed. The recent allocation of funds by Congress for adequate research in dentistry highlights the shortcomings of our present facilities and the need for additional personnel to work in this field. Some research should necessarily be basic in nature whereas other work might well have immediate clinical application. In either event, sufficient funds are available for deserving projects and should be utilized in working toward the solution of the many pressing problems that we face.

The correlation between the heavy use of alcohol and tobacco in development of cancer of the mouth which oral surgeons have noted clinically certainly should be corroborated in the laboratory. Such research could easily provide valuable clues to nutritional and chemical states that may be extremely important in the generation of malignant disease. The effect of condylar fractures on growth and development of the mandible is another important project that deserves early attention. Further study of the uses of preserved bone in surgery of the jaws, investigation of the many varied problems of cleft lip and palate, the development of more efficient tranquilizing agents and of agents that would completely control transient bacteremias are but a few examples of the many problems that require early study.

There has been little interest in basic research among oral surgeons in the past. The time has now arrived, however, when a definite program must be evolved if we are to assume our proper responsibilities and occupy a position of justified respect in the future. Such a program should be sponsored, developed and supported by the American Society of Oral Surgeons in cooperation and consultation with the American Dental Association and the National Institute of Dental Research. It must be guided by men who are sufficiently prepared and dedicated to insure its steady growth and development so that in the future we can take pride in having done our part in making the world a better place in which to live.—*Fred A. Henny.*

Honorary doctorate presented to Dr. Thoma (left) by Professor Tondury (right)

Switzerland honors Dr. Thoma

THE UNITED STATES OF AMERICA, throughout its history, has been fortunate to receive as adopted sons many great men from other countries. One of these, Kurt Hermann Thoma, came to the United States from Switzerland to study dentistry. Since his graduation with honors from Harvard University Dental School in 1911, Dr. Thoma has been an honor to the dental profession and has become one of dentistry's outstanding men.

His many contributions in oral pathology and oral surgery have been acknowledged by a grateful profession. Dr. Thoma has received numerous awards from American dentistry.

This year, the country of his birth reminded him that it, too, is not unmindful of his work. On June 1, at the opening of the Seventieth Swiss Odontologic Congress, the Medical Faculty of the University of Zurich conferred on Dr. Thoma an honorary doctorate degree "in recognition of his distinguished services in the advancement of the pathology and surgery of the oral organs by investigation and instruction." The degree was presented by Prof. G. Tondury, dean of the medical faculty.

American dentistry takes this occasion to pay its respects to the Medical Faculty of the University of Zurich for the signal honor conferred on Dr. Thoma, one of America's adopted sons and one of oral surgery's most distinguished members.

■ Carcinoma of the lower lip is seen much more frequently in men than in women. It usually follows repeated excessive exposure to the sun. As a result, men who work out of doors in sunny climates are predisposed to this affliction. The use of actinic filtering ointments and lotions may be of considerable benefit if used with regularity, but a patient exhibiting early signs of degeneration of the extraoral vermilion should be cautioned to stay out of the sun as much as possible and especially to avoid exposure to the sun while in the water since considerable radiation is reflected from its surface.

■ Early jaw dilatation and exercise after mandibular condylectomy for ankyloses produce better results than when only passive motion is employed. This treatment offsets a tendency to fibrosis and subsequent restriction of jaw motion which may occur in the region of surgery. Forcible dilatation reduces the amount of constriction and thus produces superior results. Dilatation is best accomplished by the use of a side action mouth prop used twice daily; the opening is increased to the point of tolerance each day and is kept in this position for five minutes.

■ Infections of dental origin are most active and virulent in spring and fall. This influence of the changing seasons is not clearly understood but apparently is based on the ability of the organisms to reproduce more rapidly under desirable circumstances of weather and humidity. Although this observation is primarily of academic interest, it also provides a note of caution for dealing with regions of subacute infection. Under such circumstances, a more conservative approach to the problem may be indicated.

■ Obstruction to salivary flow may be caused by production of mucous plugs in a region of stenoses in the duct system of the gland involved. This condition can best be managed by dilatation of the duct. Dilatation is accomplished by the use of probes of increasing size and the massaging of the gland in a forward direction several times daily. This procedure is most effective if done shortly before eating to insure free passage of the saliva during the period of greatest salivary activity. The possible presence of salivary calculi should of course always be ruled out by careful roentgenographic study. However, minute calculi can often be displaced forward in the main duct by massage, which makes removal possible.

■ Elixir of phenobarbital is an excellent preoperative sedative when only mild sedation is desired for an ambulatory patient. The adult dosage is 3 to 4 drams containing ¾ to 1 grain of the active drug. It should be given approximately 30 minutes prior to the beginning of the operative procedure. The drug is rapid-acting and not depressing but is not adequate when heavy sedation is required because of the patient's extreme anxiety.

Readers are invited to send their clinical observations to the editor, Fred A. Henny, Henry Ford Hospital, Detroit.

■ Suppuration from Slow Healing Sockets

Q.—What processes may be responsible for suppuration from sockets after extraction, and what procedures are indicated for this complication?

A.—Among the possibilities for this condition are the existence of residual regions of infection, the presence of foreign material in the alveolus and the presence of sequestrating necrotic bone. Sequestrated bone is the most frequent cause and is usually accompanied by excessive pyogenic granulation tissue formation. Roentgenograms may be helpful in demonstrating the presence of foreign bodies or necrotic bone fragments. Exploration with the removal of the granulation tissue is required, and the suppurative process must be traced to its source. Drainage should be maintained in the region after the débridement. Careful evaluation of the region and a check of the systemic response to the condition are suitable precautions in differentiating this localized type of response from osteomyelitis.

■ Technic in Flap Reflection

Q.—How may I improve the reflection of flaps to gain access to the alveolar process when there is difficulty in detaching the periosteum around partially erupted third molars and in regions of chronic periodontal disease?

A.—These zones are very adherent and do not respond to the periosteal elevator alone. While reflection of the flap margin is started with a periosteal elevator, the adherent fibrous tissue may be incised down to bone with a scalpel. This additional sharp dissection of dense fibrous tissue away from bone will prevent excess tension and tearing of the flap margins.

■ Removal of Impacted Upper Cuspids

Q.—What practical technical problems may be encountered in the removal of an impacted upper cuspid?

A.—Definitive roentgenographic localization is of utmost importance in guiding the surgical approach for greatest access to the impaction. Close proximity to the roots of adjacent teeth calls for caution in the use of cutting instruments and in directing the force applied by elevators. Sectioning of the crown of the impacted cuspid often is necessary. One should be prepared for the common sharp curvature of the apical third of the root which often may not be demonstrated clearly in roentgenograms.

■ Temporomandibular Joint Dislocation

Q.—If the mandible is dislocated during general anesthesia but is successfully reduced, is there any indication for further treatment?

A.—Because of the trauma inflicted in the temporomandibular joint region, provision must be made for a period of limited function. An elastic type Barton bandage for 48 hours and a modified liquid and soft diet for ten days will limit excursions of the mandible to facilitate repair of damaged joint structures. Analgesics and local heat may be necessary if pain is severe. These precautions are directed to the prevention of subluxation and chronic joint derangement.

This section devoted to queries and comments from readers on topics of current interest to oral surgeons and general dental practitioners is edited by James R. Hayward, University Hospital, Ann Arbor, to whom readers are invited to send their communications. The answers, prepared by competent consultants, do not necessarily represent the opinions of any official scientific organization. Your participation in this section is invited. The Editor

■ ENCYCLOPAEDIA BRITANNICA. William Benton, publisher. Walter Yust, editor. 24 volumes. 24,000 pages with 15,181 illustrations, charts and maps. Index. Appendix. Chicago, Encyclopaedia Britannica, Inc., 1956.

The *Encyclopaedia Britannica,* the world's greatest single source of information and knowledge according to its publisher, was founded in Edinburgh, Scotland, in 1768. The thistle symbol on its covers recalls its Scotch ancestry. The first set, consisting of three volumes, 2,670 pages and 160 copper engravings, was completed in 1771. The current 1956 edition consists of 24 volumes, 24,000 pages and about 15,000 illustrations.

Some 5,000 contributors, among them the American Dental Association's H. Trendley Dean and George B. Denton, and an editorial staff of 350, keep pace with the constant changes in every phase of human endeavor, especially the progress in science.

Unlike the old-fashioned lexicographic reference works, the *Encyclopaedia Britannica* is not a dull source of information. Its use of pictures to convey knowledge adds to its interest.

Throughout its 188 years of existence, *Britannica* has acquired a reputation for accuracy. Critics of *Britannica* usually take exception, not to what is said or how it is said, but rather to what is omitted. Dentists, for instance, may criticize the fact that all matters concerning dentistry appear combined in a single narrative, and that dental topics such as endodontics, orthodontics, pedodontics, periodontics, prosthetics, oral and plastic surgery, and operative dentistry do not appear as specific entries. Newer dental treatments, inventions and procedures such as fluoridation are hardly mentioned.

An encyclopedia, however, is not a dictionary; the essential difference between these reference works is that a dictionary attempts to define the meaning of each term listed where an encyclopedia concentrates all available facts concerning an individual field into constantly revised and rewritten specific articles, treating the whole subject as a unit.

Each year the *Encyclopaedia Britannica* is reprinted and each annual reissue contains revisions which are deemed most essential. In less than 20 years, there have been 25 of these new printings. As many as 3,000,000 words of text and hundreds of illustrations are changed in each printing to bring *Britannica* up to date. The result is that the set each year is as abreast of the time in style, information and knowledge as it is humanly possible to make it.

The *Britannica Book of the Year 1956* was also received. It is an excellent compilation of factual records of great events of the year 1956.—*Paul H. Glucksman.*

■ BIBLIOGRAPHY OF MEDICAL REVIEWS: 1955. Edited by Seymour I. Taine for the Armed Forces Medical Library. 74 pages. Washington, D. C., U. S. Government Printing Office, 1956.

In recent years there has been a gradual increase in the review type of article in science in general and medicine in particular. The review article follows the abstract journal as a means of helping the practitioner keep up with advances in his field. Reviews vary in length from a few pages to hundreds of pages. The average review constitutes a survey of the latest advances in a specific field.

The present publication came into being when the Armed Forces Medical Library decided to compile the bibliography as a by-product of its *Current List of Medical Literature* operation.

The bibliography covers about 350 publications, of which only seven are dental journals. Reviews are listed in the fields of clinical and experimental medicine and the allied biomedical fields such as dentistry (13 articles),

pharmacy, nursing and psychology. The reviews are grouped alphabetically by subject headings. All the articles selected appeared in 1955 issues of periodicals.

"This initial effort," says the editor in a preface, "serves generally as a trial balloon, and comments on all aspects of the venture are earnestly solicited."

The concept behind the bibliography is sound, but if an annual bibliography of reviews were to cover all fields of medicine thoroughly, it would have to be much larger than this initial work. The volume's coverage of dental review literature is so meager as to be all but valueless.

The material is arranged for easy reference. It is printed by the photo-offset method in a pleasing style and bound in soft covers.—*N. C. Hudson.*

■ CANCER—A MANUAL FOR PRACTITIONERS. Third edition. 321 pages. Index. $2. Boston, American Cancer Society (Massachusetts Division), 1956.

The 50 chapters of this book by as many authors cover the present knowledge on the subject of cancer authoritatively and concisely. The first nine chapters give brief and precise statements on the history, epidemiology, pathology and diagnosis of cancer in general and something of the present research in this field. The next 30 chapters give specific details regarding the etiology, diagnosis, treatment and prognosis of malignancies of the various tissues and organs of the body.

Although the book is intended for medical practitioners, most of the chapters are written in a simple and direct style which might be read with profit by the layman.

The chapters on cancer of the lips, the mouth and the salivary glands are of interest to the dentist, and one chapter on the role of the dentist in cancer control is pertinent. Most well-trained dentists will find these chapters insufficient for extending knowledge in the field and will prefer to consult more complete texts, but they will find the other chapters exceedingly interesting as a readable and authoritative review of the status of cancer diagnosis and treatment for the entire body.

Other chapters include malignant disease in children, care of the patient with advanced cancer and the program of cancer prevention and control, both local and national.

The book is factual, well written and interesting, and the eminent authors make it a thoroughly dependable source of information.—*Charles W. Freeman.*

■ SOCIAL SECURITY—FACT AND FANCY. By Dillard Stokes. 208 pages. Appendixes. $4. Chicago, Henry Regnery Co., 1956.

Since its inception, the Old Age and Survivors Insurance program, popularly known as social security, has been the subject of much controversy. In this volume, Mr. Stokes, an attorney and newspaperman, has reviewed this program and summed up the basic criticisms which have been leveled at social security over the years. A proponent of the principle of social security, the author suggests a substitute program which he claims is not subject to these criticisms.

The burden of Mr. Stokes' argument is that the social security program is not what it purports to be. He argues that, contrary to popular belief, it is not an insurance program, for an individual participating in the program has no right to any benefits. He classifies the program as one of relief, through which the community pays relief benefits to those whom it determines to be qualified for such benefits according to arbitrary statutory and administrative conditions.

The author supports his position by showing the lack of relationship between the premium or tax paid to the benefits that might be forthcoming to an individual at age 65. He examines and criticizes the fiscal management of this program and suggests that the trust fund out of which benefits are paid could become dissipated in the not too distant future. Numerous individual case histories, both actual and hypothetical, are set out in the text to illustrate the author's view that the present program is arbitrary and creates inequities. These criticisms, although answerable in part,

are worthy of the public's serious consideration.

In lieu of the present program, Mr. Stokes suggests a program in which the government issues annuity bonds to participating citizens in consideration for an annual premium of 8 per cent on the first $4,200 of an individual's annual income. This premium would be shared equally by the employee and the employer. The annuity bonds would be redeemable at age 65 for a life income measured by the value of the bonds. Under this program, every individual would be assured of a pension or of the cash value of the bonds for his estate. The author does not explain how this program would relate to the self-employed.

Mr. Stokes has set out his argument in a style which is easy to follow. His argument is a convincing one. Therefore, the reader of this volume must be cautious in reaching a conclusion as the positive side of the present program is not set out for his consideration. This volume will be of interest to those concerned with the broad objectives of our national social security program.—*Howard N. Greenblatt.*

■ THE NEUROSES IN CLINICAL PRACTICE. By Henry P. Laughlin, M.D. 802 pages with illustrations. Index. Appendix. $12.50. Philadelphia, W. B. Saunders Co., 1956.

Almost any professional member of the healing arts will find this detailed study of the neuroses, many of which are encountered in his work, interesting and of real help to the understanding of problems met in practice. Much of the book deals with the history, diagnosis, clinical features and treatment of various neuroses, including the anxiety reactions, phobias, conversion reactions including hysteria, dissociative reactions, depression, overconcern with health, obsessive-compulsion reactions, and neuroses following trauma. Discussion of the defense mechanisms occupies 80 pages.

The material is in textbook style, well organized and quite readable by anyone with an elementary knowledge of psychology. The approach is mainly eclectic, but leans toward the psychoanalytic viewpoint. Included are an extensive table of contents, glossary, index and references for each chapter. There are 32 tables and over 200 case summaries. The publisher has contributed good typography, paper stock and binding.

The book may have been written primarily as a preclinical text in basic psychiatry for medical students, but dentists, psychologists, social workers and others in the health professions will find that for either general reading or reference the author has assembled much of value from the literature and his experience. It avoids the narrow limits of any one school of thought and is neither too perfunctory nor too exhaustive.

The appendix includes an interesting outline classification of emotional and mental illness of which the neuroses constitute only a fractional part, although, on the basis of incidence, a significant part. The author should be encouraged to add other volumes in a series with this outline as the frame of reference.—*Clarence M. Peebles.*

■ Five Year End Results In Epidermoid Carcinoma of the Lip with Indications for Neck Dissection. Leslie H. Backus, M.D. and Clement A. DeFelice, M.D. Plast. & Reconstr. Surg. 17:58 Jan. 1956.

Epidermoid carcinoma of the lip is the most common malignancy of the alimentary and respiratory tracts. With the exception of basal cell and squamous lesions of the skin of the face, no head and neck neoplasm is more frequently encountered. The study discusses the indications for prophylactic neck dissection in epidermoid carcinoma of the lip. The conclusions are the result of a study based on 226 patients, 84 per cent of whom had no clinical evidence of cervical metastases on admission. Of these patients, 15.5 per cent had cervical metastases on admission and 4.8 per cent developed metastases during the follow-up period. Hayes Martin has recently analyzed 421 consecutive instances of epidermoid carcinoma. In evaluating Dr. Martin's results, it was found that prophylactic neck dissection would have been of benefit to only 6 per cent of the entire group, or a ratio of 1:16. In other words, 15 needless operations for one patient's benefit would have been the result. His analysis correlated closely with the results of the 226 instances found in the present study. There were 52 indeterminate and 174 determinate cases in the series. Of the determinate cases which were followed up closely after surgery, 146 did not have metastatic involvement on admission. Two of the patients had their neck metastases first appear contralaterally and 137 never developed metastases. This meant there were 139 patients for whom prophylactic neck dissection would not have been beneficial, leaving only seven patients for whom it might have been of value. In four of these seven patients, five year cures were obtained by therapeutic neck dissection, which left only three patients who would have been helped by prophylactic neck dissection. This was only 2 per cent of the series for a ratio of 1:49. Epidermoid carcinoma of the lip exhibits certain biological characteristics which justify treatment of the cervical nodes by neck dissection, done therapeutically rather than prophylactically, provided there is adequate follow-up of patients.—*Irwin A. Small.*

■ Results of Surgical Treatment of Cancer of the Tongue. Arthur J. Cremens, M.D. Surgery 39:49 Jan. 1956.

A follow-up study was made of patients who had been operated on for carcinoma of the tongue. The procedure advocated is the en masse removal of the primary tongue lesion, the intervening tissue and the regional lymph bearing areas. All patients with cancer of the tongue seen at the tumor clinic at the University of Minnesota since June 1953, plus the private patients of the author that were deemed suitable for surgical care, were operated on as described. Criteria of operability were as follows: (1) the lesion should be confined to one-half of the tongue or the floor of the mouth, and grossly extended beyond the midline; (2) lesions arising primarily in the base of the tongue were excluded since these are highly undifferentiated growths and are ordinarily radiosensitive; lesions arising in the middle third of the tongue and extended posteriorly were included and some resections were carried as far posteriorly as the epiglottis; (3) clinical evidence of regional lymph node involvement was not looked on as a contraindication unless the nodes were massively enlarged, agglutinated, and fixed; (4) age of the patient was no contraindication if general condition was satisfactory; six of the patients in this series were over 70 years of age; (5) there should be no evidence of distant metastasis. Thirty-two patients with cancer of the tongue were operated on in the fashion described; 60

patients (50 per cent of the entire group) had histological evidence of lymph node involvement. In 21 patients without palpable cervical lymph nodes, 9, or 43 per cent, showed histological evidence of lymph node involvement by metastatic cancer. Of 23 patients operated on, 15, or 65 per cent, were alive and free of disease over a period of three years. Of 10 patients operated on five years previously, 7, or 70 per cent, were alive and free of disease. The results achieved through surgical treatment of these instances of cancer of the tongue are sufficiently encouraging to warrant its continued trial and clinical application.—*Irwin A. Small.*

■ COMPLICATIONS OF FACIAL BONE FRACTURES. Robert F. Stokes, M.D. Plast. & Reconst. Surg. 17:73 Jan. 1956.

Fractures of the facial bones are frequent injuries; often they are complex. Permanent distortion of features and disabling deformities often follow improper or inadequate treatment. Complications and their treatment include the following.

Malunion: To obtain satisfactory results in the treatment of malunion, two important points must be kept in mind. Careful clinical and roentgenographic evaluation of the patient must be made. A proper method of treatment must be selected, and its effectiveness must be re-evaluated before the bones have set and while conditions are still favorable for further correction. If satisfactory progress is not being made, treatment should be modified or changed.

Injuries of the visual apparatus: These include displacement of the ocular globe and damage to bones of the orbit which can produce a displacement of the eye in any direction. The most common disturbance follows a blow to the zygomatic bone or infraorbital rim. When the orbital floor is pushed downward, the eye is carried along with it. Injuries are often overlooked until the soft tissue swelling subsides, and the patient calls attention to the deformity and diplopia.

Loss of sight is one of the most common complications of facial bone injuries. Vision may be lost if the optic nerve is injured by a fragment of bone, or the eyeball itself may be injured by a bony spicule.

Obstruction of the nasal lacrymal duct: The nasal lacrymal duct is 17 mm. long and only 4 mm. wide. When a fracture of the maxilla involves the canal, the duct is apt to be injured. If stenosis follows, and the lacrymal sacs become infected, the patient develops swelling near the inner canthus on the side of injury. Sometimes the obstruction is temporary, and the condition improves spontaneously.

Nerve injury: Anesthesia is a frequent occurrence when a branch of the fifth cranial nerve is injured during a facial bone fracture. Fortunately, the loss of sensation usually is temporary. In the great majority of instances of neuroma, the effect of the injury on the nerve is transient. There have been instances when a painful neuroma had to be treated by excision of the nerve at the infraorbital foramen. The permanent destruction of a sensory nerve produces not only a disagreeable anesthesia but predisposes the region to the development of trophic ulcerations. When facial paralysis occurs, injury of the seventh cranial nerve or one of its branches might easily pass unrecognized for the first few days after injury. This could happen because swelling of the soft tissue interferes with normal muscular activity. When the swelling subsides, the region should be examined carefully for the return of muscular function.

Respiratory obstruction: Obstruction may result from hemorrhage. When blood from the inside of the nose or mouth runs into the throat, it may seriously interfere with the passage of air to the lungs. Danger that this may occur is serious when the patient is unconscious and is allowed to lie on his back.

Relaxation of the tongue also may cause respiratory obstruction. Fracture of the mandible and nearly all those in which the central third of the bone is displaced posteriorly may interfere sufficiently with control of the tongue so that it falls back against the posterior wall of the pharynx where it partially or completely obstructs inspiration. The simple procedure of putting a suture through the tongue and applying forward traction will relieve the obstruc-

tion. Tracheostomy is necessary if additional soft tissue injury or other complications preclude fixation of the mandible.

Bony occlusion of the airway results from depressed fragments of nasal bones. Fractures of the maxilla are not among the common causes of respiratory obstruction but can seriously occlude the upper air passages. One of the most unusual complications is the result of a severe displacement of the maxilla which forces the palatine bones backward against the posterior pharyngeal wall to obliterate the nasopharynx.

Restriction of mandibular motion: It is not unusual for fractures of the mandible to be followed by a period during which motion of the jaw is limited. Limited motion usually results from atrophy of the muscles of mastication which has developed during immobilization. Additionally, when the fractured zygomatic arch is depressed against the coronoid process of the mandible or the insertion of the temporalis muscle, the patient will have pain and difficulty in opening his mouth.

Fibrous ankylosis is often called false ankylosis and is due to the development of strong fibrous adhesions in the soft tissue and muscle attachments that attach to the coronoid process. The treatment varies with the extent and the location of the adhesions. Sometimes mandibular motion can be restored, first by dilation of the mouth when the patient is under general anesthesia, and then by the use of a mechanical device to keep the mouth open. Sometimes excision of the scar bands and use of a dilating appliance is of benefit. Some patients require the removal of the coronoid process. Condylar fractures accompanied by a dislocation of the condyle may be followed by ankylosis of the temporomandibular joint. This condition apparently occurs with less frequency than is commonly believed. Resection of the involved bone or creation of a false joint may be necessary to restore mandibular function.

Vascular injury: The face has a rich blood supply, so that vascular injury sometimes complicates facial bone fractures. Hemorrhages or hematomas are seen frequently. They are serious but seldom are the cause of diagnostic difficulties. Arteries that lie close to bone can easily be injured by bony spicules, and a true aneurysm may develop at the site of damage of the vessel wall. The rate at which it develops depends on the extent of injury to the outer coats of the artery. Many months may elapse between the time of trauma and development of a recognizable aneurysm. Arteriovenous fistulas are unusual complications of injury. Most of those described about the face and scalp have been classified as congenital types. History of trauma and arteriograms may be helpful in establishing the nature of the lesion.—*Irwin A. Small.*

■ JAUNDICE DURING CHLORPROMAZINE (THORAZINE) THERAPY. Stewart Lindsay and Richard Skahen A.M.A. Arch. Path. 61:84 Jan. 1956.

Chlorpromazine (Thorazine) is currently being administered to patients for a variety of diseases. In addition to its use in psychoneurotic and psychotic disorders, this drug has also been used for its antiemetic, analgesic and sedative effects. One complication of this therapy is jaundice, the reported frequency of which varies from 1.4 per cent to 4.8 per cent. Although a number of reports have described hepatic lesions in patients developing jaundice after chlorpromazine therapy, the pathogenesis and evolution of these lesions are obscure.

Some attribute these lesions to a hypersensitivity to the drug, and it is known that jaundice appears more frequently in patients receiving higher doses for longer periods. Others, however, are impressed by the inflammatory reaction of the lesion and regard the lesion as probably the result of a direct toxic injury to the hepatic cells, although this injury appears to be extremely mild.

The clinical and laboratory records and descriptions of the histologic lesions of the livers of five patients who developed jaundice after administration of chlorpromazine are presented. Hepatic lesions disclosed by biopsy were different and easily distinguishable from those occurring in external biliary obstructions, from various other forms of severe toxic hepatitis and from cholangiotic biliary hepatitis or

cirrhosis. The cellular injury is mild and reversible and principally centrilobular. Bile stasis is confined to the central biliary canniculi. The hepatic lesions which may result from the administration of chlorpromazine can be differentiated in needle biopsies of the liver from those associated with external biliary obstruction.—*Irwin A. Small.*

■ PHYSIOLOGIC BASIS OF TRANSFUSION THERAPY IN HEMOPHILIA. Kenneth M. Brinkhous, George D. Penick, Robert D. Langdell, Robert H. Wagner and John B. Graham A.M.A. Arch. Path. 61:6 Jan. 1956.

It has long been recognized that blood transfusions have controlled the hemorrhagic crisis of hemophilia and those hemophilia-like diseases which are called the hemophiloid states. Lane, in 1840, probably was the first to employ blood transfusions for control of hemorrhage in a patient with a bleeding diathesis. Since about 1910, transfusions have been given for control of hemorrhage with increasing frequency, until today they are commonplace. The development of a therapeutic regime has been largely empirical, since the pathologic physiology of hemophilia has been poorly understood until recently.

Only within the last 20 years has there been general acceptance of the thesis that the essential defect in hemophilia lies in the plasma rather than in the formed elements or in the blood vessel wall. Today most investigators agree that true hemophilia results from the congenital deficiency of a specific plasma protein, antihemophiliac factor (AHF), which is needed for a normal operation of the complex coagulation mechanism. The severity of the disease is relatively constant in a given family and is dependent on the degree of AHF deficiency. The beneficial effect of transfusions in promoting hemostasis is dependent upon the AHF contained in the bone or plasma.

The rationale of the program of plasma transfusions in the treatment of hemorrhagic crises in the hemophiliac is outlined. Factors considered are: (1) sources of AHF for transfusion, (2) minimum hemostatic levels of plasma AHF, and (3) the rate of loss of transfused AHF. Sources of AHF are normal plasmas or plasma fractions. Normal plasma from different subjects varies widely in AHF content. In stored bank blood or frozen plasma, approximately half the initial AHF content is lost in two weeks. Lyophilized plasma has about half the potency of fresh plasma. AHF in reconstituted fraction I is about one third of the mean's normal value.

Fresh plasma from selected donors appears to be the best source of AHF currently available. The minimum hemostatic level of AHF may be as low as 5 per cent of normal, although some data suggest that it may be 15 per cent to 20 per cent of normal or higher. Of the simple laboratory tests used to follow AHF levels after transfusions, the partial thromboplastin time and the thromboplastin generation tests appear most suitable for following AHF changes in the critical range. Thus utilization of AHF by hemophiliacs after transfusion appears to be rapid and exponential. In human hemophiliacs the half life of injected AHF appears to be about four hours; in canine hemophiliacs about two hours. These observations explain the need for frequent transfusions. Delayed or inadequate treatment may result in dissecting hematomas and tissue necrosis with greatly increased requirements for AHF.—*Irwin A. Small.*

Announcements

■ Oral Surgeons Install G. T. Gregory as President

G. T. Gregory of Indianapolis was installed as president of the American Society of Oral Surgeons at its September 25-28 meeting in New York City. Chosen president-elect was W. T. Foster of San Antonio, Texas. Completing the roster of officers are: F. F. Pfeiffer of New York, vice-president; F. H. O'Halloran of Evansville, Ind., and E. C. Thompson of Urbana, Ill., council members, and J. R. Hayward of Ann Arbor, Mich., member of the American Board of Oral Surgery. The society's council reappointed Frank B. Hower of Louisville, Ky., secretary-treasurer, and Miss Ruth Whalen of Richmond, Va., executive secretary.

■ Denmark Honors American Oral Surgeon Harold Krogh

Harold W. Krogh, oral surgeon of Washington, D. C., and past chairman of the American Dental Association's section on anesthesiology, has been awarded the Knight's Cross of the Royal Order of Dannebroge by his Danish Majesty King Frederik IX. Dr. Krogh, who has lectured and taught in Scandinavia, and who is of Danish descent, was cited for his services to Denmark.

■ Paul J. Flory of Cornell to Head Mellon Institute Research

Paul J. Flory, acting chairman of the department of chemistry at Cornell University, has been chosen Mellon Institute's executive director of research. Dr. Flory has pioneered research on the constitution and properties of rubbers, plastics, fibers, films and proteins and is the author of 106 papers in scientific journals. He is the author of *Principles of Polymer Chemistry*, published in 1953. Dr. Flory is a member of the National Academy of Sciences, which is composed of leading scientists of the United States. He holds membership also in the American Chemical Society and in the American Association for the Advancement of Science.

■ Scientific and Vacation Program Combined in Pacific Coast Conference

A scientific program has been combined with vacation attractions in the Tenth Triennial Pacific Coast Dental Conference to be held in San Diego August 19-22. The Southern California State Dental Association, host to the conference, reports that some of the nation's foremost clinicians will participate in the program. In addition, trips are planned to the colorful Mexican city of Tijuana and to points of interest in San Diego. Reservations may be addressed to C. W. Gilman, secretary, 219 E. 8th St., National City, Calif.

■ Two Year Oral Surgery Course Offered at Illinois

Applications are now being accepted for a two year, full-time course in oral surgery at the University of Illinois College of Dentistry. The course, which begins September 1957, carries credit toward a Master of Science degree. Further information may be obtained from Daniel M. Laskin, department of oral and maxillofacial surgery, at the school in Chicago.

■ Tufts Offers Courses for Oral Surgeons

Three courses for oral surgeons are offered by Tufts University School of Dental Medicine in Boston. Oral pathology, to be taught Thursday afternoons for eight weeks beginning February 7, is under the direction of Irving Meyer, Gerald Shklar and Irving Glickman. The clinical, roentgenographic and microscopic aspects of oral disease will be presented. Laboratory sessions are planned. Fred Henny, chief of the dental department of Henry Ford Hospital in Detroit, lectures on oral surgery April 25-27. Dr. Henny will discuss diseases of the salivary and lymph glands, the temporomandibular joint, fractures and infections. Modern concepts of drug therapy also will be covered. The third course, intravenous anesthesia for the dental office, is offered March 25-29. Harold Krogh of Washington, D. C., and Adrian O. Hubbell of Long Beach, Calif., will discuss the pharmacology of the barbiturates and the physiology of intravenous anesthesia.

■ Harry Lyons of Virginia Installed as Association President

Harry Lyons of Richmond, Va., widely-known dental educator, was installed as president of the American Dental Association at the close of its 97th annual meeting October 4 in Atlantic City, N. J. Dr. Lyons, dean of the School of Dentistry of the Medical College of Virginia, has served on the school faculty since his graduation in 1923. He is also professor and head of the department of oral pathology, diagnosis and therapeutics, and of periodontics.

In addition to his activities in the field of education, he has been prominent in affairs of the Association. He served as speaker of the House of Delegates from 1952 to 1955. He was chairman of both the Special Committee on National Emergency Dental Service and the Nomenclature Committee. For 17 years, he was a member of the Council on Dental Therapeutics.

Dr. Lyons has been a leader in national efforts to obtain substantially increased support for dental research. He is a member of the National Advisory Dental Research Council of the National Institute of Dental Research and formerly served a two year term on the Committee on Dentistry for the National Research Council.

■ World Dentistry to be Reviewed at International Congress

Reporters and co-reporters are studying and summarizing papers on dental subjects from all over the world, preparatory to presenting these condensed reports to the F.D.I. International Dental Congress in Rome September 7-14, reports Obed H. Moen, F.D.I. treasurer for the United States.

The F.D.I.'s *International Dental Journal* has exclusive rights for the publication of the reports, Dr. Moen states. The first preliminary report appeared in the September 1956 issue, and publication will continue through July 1958.

Members of the F.D.I. receive copies of these reports. Those who have been members through 1956 and 1957 are entitled to a 50 per cent reduction in the enrollment fee for the international meeting.

Information on F.D.I. membership and on the Congress may be obtained from Dr. Moen, 6 Main St., Watertown, Wis., or from Harold Hillenbrand, secretary, American Dental Association, 222 E. Superior St., Chicago 11.